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LIX

LEE WALLACE DEAN, SR.,

A PERSONAL APPRECIATION

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ST. LOUIS, MO.

In dedicating this issue of the ANNALS to the memory of its late Editor, Lee Wallace Dean, Sr., the Staff and some of Doctor Dean's colleagues and students who have contributed articles, seek in a small measure to express their respect and affection for him.

If this short sketch of my association with Doctor Dean takes slight notice of his dates, his honors and his degrees, it is because these things were the least important part of him. His own outlook was upon other more immediate matters, which taken alone might have been mistaken for the trivia of a busy life, but which seen together built up a career of force and influence and service and culminated in that personality known to us as The Chief. With complete lack of originality this title is conferred on almost any departmental head. Only occasionally it carries weight. This one did.

My earlier acquaintance with him was the casual, conversational one which springs up at medical meetings, but our mutual interest in nasal research drew us together, even before the death of Doctor Hanau W. Loeb in 1927 united our efforts as co-editors of the ANNALS.

In the fall of the same year Doctor Dean came to St. Louis to head the Department of Otolaryngology in Washington University. He was destined not only to build out of chaos a teaching group of the first order but also to lay down a pattern of organization and productive method in human relations that was to have a deep and significant influence upon the lives of those of us who came in daily contact with him. It might be said that his predominant characteristic was an amazing ability to so innoculate his friends, colleagues and subordinates with his plans and ideals as to make them constantly conscious of him as a power with whom they had hourly to deal and at the same time a rock on which to anchor in any storm.

The house which he came to set in order was something of a mansion, designed by Greenfield Sluder in the grand manner, but now in fair disarray partly as the result of Sluder's easy housekeeping and partly owing to the sad incapacity of that genius' last years. Like many another brilliant mind Sluder devoted himself with religious fervor to the things which attracted him and avoided the rest either with a pained and childlike helplessness or a gay indifference. His staff was imbued with his zeal for original observation and scientific honesty and with some of his passion for detail, but the Department was forever falling apart from lack of any vestige of cohesive organization.

Whether by inspiration or accident, precisely the right man was chosen to take charge. The sagacity of McKim Marriott, then Dean of the Medical School and one of the ablest administrators, pointed the way. Dean and Marriott continued to entertain the warmest regard for one another and though they sometimes sparred over details, they never fought over principles.

The skill with which the new Chief welded all the warring parts into a harmonious whole was a model of human insight. With his tact he buffed the rough edges of personal animosities. With his endless queries he conveyed the idea that each individual was expected to be doing something a little beyond what he had been doing yesterday. With studied patience he taught that the Department transcended the individual, and that the welfare of the patient transcended everything else. He breathed, and ate and slept and dreamed Department. He planned and strove and fought for Department. Sometimes I thought he even hunted and fished Department but there are those who deny this.

At any rate there blossomed through his efforts a group of investigators and clinicians, full and part time, who worked together,

who shared their ideas in the laboratories and corridors and seminars without jealousy or reserve, whose early investigations received such responsive respect and recognition that they were perpetually keyed up to greater efforts, who finally learned to collaborate as a pliable effective and vital organization.

Laboratories and technicians were always available. Apparatus materialized. The Chief spent days and tore what hair he had over the budget, but there were always funds. The woods abounded with anonymous donors. His enthusiasm was quiet but so insidious and infectious that his friends caught it and wrote checks.

His eyes were constantly on the horizon. His acquaintance with the country's educators was personal and intimate. He knew what was being done in this state board and that medical school and that Professor Smith of Jones University was unhappy—and did we want him in our Department.

He had unlimited faith in the human mind, but none whatever in the human memory. He could scarcely ask anyone to pass the butter without sending him a written memorandum of the conversation with a request for an acknowledgment. These acknowledgments he filed *in extenso* to his eternal satisfaction and the confusion of any, great or small, who fumbled their engagements, promises, or obligations. He was never trivial and most certainly never exultant about it. He was quietly—and so effectively—protecting his beloved Department.

He had not been with us long before this thing began rubbing off on the rest of us. The Thursday Morning Conferences did it. These were the most competent spontaneous discussions I have listened to. Even the neophytes knew better than to present a case incompletely prepared. Even the Staff tried to talk sense. But what lifted this Thursday Morning above the hundred other similar conferences was the rich fund of experience of Doctor Dean himself and his uncanny ability to remember the details of his cases, seen years before. At first I entertained the cynical notion that some of them were apt inventions of the teacher driving home his point, but as time went on I heard them repeated not only without variation, but with trimmings by Bill Johnson or Al Cone, who had examined the poor patient in their sainted Iowa and usually remembered his name! I would give much to recapture the flavor of those Thursday Mornings.

Having thus put his house in order, he launched with great vigor, and his usual singleness of purpose, upon the organization of

the eight-months' graduate course which drew thirty students each year from all over the United States and covered itself with distinction. It set out with a staff of some thirty members, demonstrating both the basic and the clinical subjects and combining the research of the two. Today the teachings and ideals of this group pervade many an office, sick bay and Army camp—a source of deep satisfaction to the Chief in his days of retirement.

Dr. Dean was never troubled by any division of interest between his Department and his family. He rolled them all into one. No man was more dependent upon his spouse, and happily so. They did not move, one without the other. They worked and played together. They traveled and attended medical meetings together. They farmed and they hunted and they fished together. Week after week they shuttled back and forth together between St. Louis and their old home in Iowa City to which their fond attachment held them through the years. Their roots were in Iowa and there remained. And as they lived together, so together they died, in the same year.

Their friends and colleagues and a host of students will always retain a deep affection for this pair.

Doctor Dean was born in Muscatine Iowa, March 28, 1873, and died in St. Louis February 9, 1944. During his long span of years he was Professor of Otolaryngology, Head of the Department, and Dean of the Medical School in the University of Iowa, and later Professor and Head of the Department in Washington University, St. Louis; he was President of all the national otolaryngological societies in turn, a member of the American Board of Otolaryngology, and Co-Editor of the ANNALS. In 1937 he was honored with the deRoaldes Medal of the American Laryngological Association.

A life as full as any man could wish.

LX

BILATERAL ATROPHY OF THE INTERNAL CAROTID ARTERY. A RARE ANOMALY.

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BOSTON, MASS.

Routine collection of human temporal bones with microscopic examination of this autopsy material frequently yields data of unanticipated significance. At a recent autopsy in the Massachusetts General Hospital permission was obtained to remove the petrosae from a 48-hour-old white male infant.

The summary of the clinical history stated that this premature infant was delivered during the eighth month of pregnancy following labor of apparently normal type and duration. At birth the infant was quite cyanotic but no other abnormality was noted. The child did poorly after delivery and breathed with a gasping type of respiration. The breath sounds were never adequate over the chest and continuous oxygen atmosphere was maintained. In spite of this the child gradually became increasingly cyanotic and died 48 hours after birth.

Necropsy was performed by Dr. B. Maisel. An abstract of his report follows: *Anatomic Diagnosis*, prematurity and incomplete expansion of lungs. The body measured 42 cm. over-all and 34 cm. from crown to rump and weighed 1940 gm. The lungs showed almost complete collapse of all lobes. The lingular portion of the lower portion of the right upper lobe was partially aerated. Sectioning disclosed a firm red parenchyma. Microscopic examination of the lung showed that the alveoli were almost completely collapsed and the interalveolar vessels were markedly congested. The heart was observed to be more pointed than usual. There was a widely patent foramen ovale that was not occluded by any septum. The ductus arteriosus was widely patent. It was stated that the neck vessels were normal in their origin and distribution. The brain weighed 130 gm. The subarachnoid vessels were markedly congested but there was no hemorrhage about the brain or in the brain. The gyri and sulci were normal.

From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

The microscopic sections of the petrosae were prepared in the Mosher Laboratory of the Massachusetts Eye and Ear Infirmary. The sections were cut in horizontal plane and stained with hematoxylin and eosin stain. Study of the serial sections revealed, as may be seen in the accompanying photographs, atrophy of the internal carotid artery at the lower level of the petrosa. The condition was bilateral.

In each petrosa at the level of the midmodiolar region of the cochlea (Figs. 1 and 2) the arterial wall was collapsed. On the right a few delicate strands of endothelial cells crossed the collapsed lumen suggesting that the lumen may never have been properly patent here. At a lower level, however, red blood cells and a few leucocytes were trapped within the lumen. Descending in the serial sections in each ear the calibre of the vessel decreased and the arterial wall exhibited an involution resulting in more or less complete obstruction of the lumen as shown in Fig. 3. At a still lower level the vessel disappeared entirely as shown in Fig. 4, where only a few muscle fibers indicated the location of the atrophic vessel. At the superior level of the petrosa the vessel was patent and blood cells appeared within the lumen. The pericarotid nerve plexus was normal and did not encroach upon the vessel wall at any observable point.

On the right, two aberrant vessels were present in the cochlea. One of these is illustrated in Fig. 1 and appears in the scala media of the apical turn. The other, not shown in the photographs, travelled along Reissner's membrane for a short distance. Other than these two aberrant vessels, no further abnormalities were noted in the petrosae. Ossicles, ossicular muscles, and inner ear structures were normal in each ear.

PERTINENT LITERATURE

A survey of the literature has thus far revealed only one other case of absence of both internal carotids. This was Fisher's[14] case of a 39-year-old man who died of cerebral hemorrhage. No symptoms were present during life although the condition admittedly may have hastened the end. The basal artery was in this case found to be double its normal caliber.

Orr[21] in 1906 found a low division of the common carotid with a very small internal carotid which was only 2 mm. in caliber. This was observed grossly in the dissecting room on a well-developed adult male. The arteries supplying the brain were normal in size.

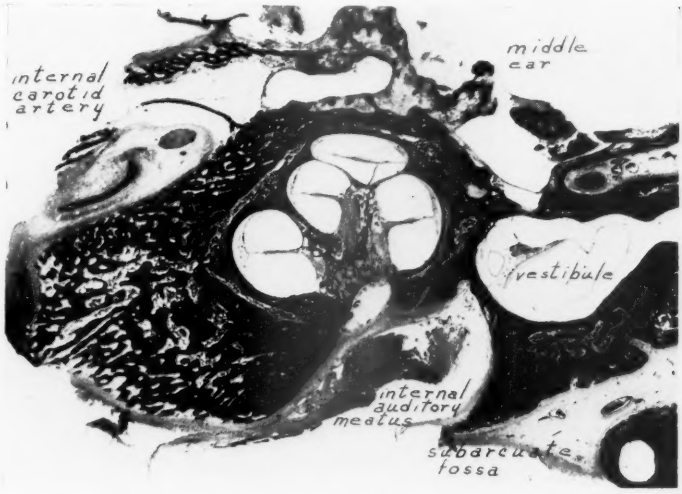

Fig. 1.—Right ear showing collapsed internal carotid artery at the level of the cochlea. Note also the aberrant vessel in the scala tympani of the apical turn of the cochlea.

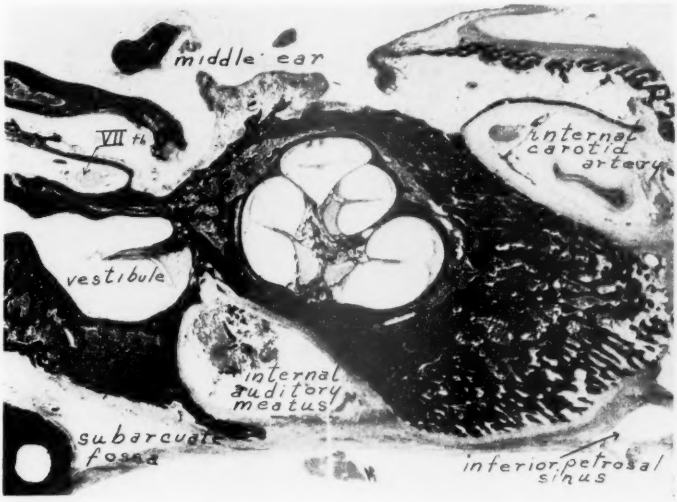

Fig. 2.—Left ear showing internal carotid artery not quite so completely collapsed at this level as on the right but obviously not normal.

Poynter[23] lists as his eighth and last example in Group IV among *Irregularities in the Development of the Aortic and Pulmonic Arches, etc.*, under heading D, "Abnormal Obliteration or Persistence of Segments of the Arches or Dorsal Roots." He states:

> "Obliteration of the Third Arch and the Roots Beyond. This condition is characterized by the absence of the internal carotid; the condition may occur on either the right or left side . . . All of the following cases are a failure of development on the left side except that of Todd which is on the right side and Fisher which is on both sides. Todd (1787), Koberwein (1810), Quain (1844), Peugnet (1876), Wyeth (1878), Flemming (1895), Fisher (1914)."

Ask-Upmark[2] classifies malformations of the internal carotid into three groups: (1) absence of the artery, (2) abnormal origin of the artery and (3) tortuosity of the course of the artery in the neck. He emphasizes particularly the importance and significance of the carotid sinus. This bulb-like dilatation of the internal carotid is located at the point of origin of the internal carotid artery from the common carotid. This organ possesses within its walls mechanism for control of blood pressure, heart beat, adrenalin output and respiratory rate. External pressure on this region will alter the pulse rate and in highly sensitive individuals may cause death.

Ask-Upmark also calls attention to the fact that in cows there is no internal carotid, the brain being supplied by branches from other arteries, most important of which is the occipital artery.

Besides the particular anomaly above described which is rare in the human subject, other anomalies of the internal carotid artery on one or both sides have occasionally been reported in the literature.

Quain[24] described an absent internal carotid artery whose place was taken by two branches from the internal maxillary which entered the cranium through the foramen rotundum and foramen ovale respectively. From their junction the ophthalmic was given off. In the 1882 edition[25] of his work Quain stated that a few examples of the entire absence of the internal carotid artery have been reported.

Curnow[11] described bilateral recurrent branches of the internal carotid within the skull going to the posterior fossa.

Stimson[28] observed a loop in the artery just above the bifurcation of the common carotid.

Batujeff[4] reported the origin of the basilar artery from the internal carotid in a 25 to 30-year-old man. The caliber of the left internal carotid was 10 mm.; that of the right, 6 mm.

Fig. 3.—The involution of the carotid wall. The vessel is of smaller calibre than normal for this level which is at the basal turn of the cochlea. Right.

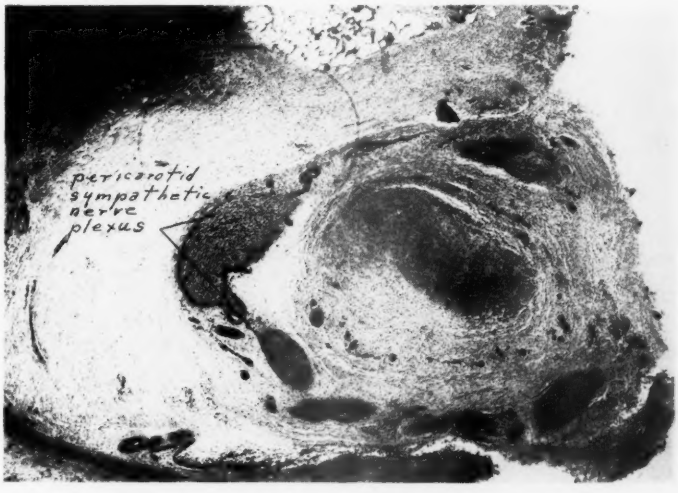

Fig. 4.—At the level of the curve of the posterior inferior semicircular canal the artery has no lumen and consists of a mere wisp of a few muscular fibers. The condition was bilateral.

Shepherd[30] stated that the internal carotid artery has been known to be absent, its place being taken by the artery of the opposite side or by a branch of the internal maxillary. The ascending pharyngeal, occipital, lingual, or transverse facial arteries may arise from the internal carotid. A large communicating branch from this artery has been seen going to the basilar artery while in the cavernous sinus. The posterior cerebral not infrequently comes off from one of its branches, the posterior communicating.

Smith[26] reported an anomaly of the internal carotid in an Egyptian subject in which the internal carotid on the left side, after emerging from the carotid canal, gave off a branch 4 mm. in diameter which passed along the inner side of the Gasserian ganglion, perforated the dura and anastomosed with the basilar artery at the level of the insertion of the trigeminal nerve into the pons.

Kantor[17] described and illustrated two cases of deep division of the internal and external carotid with almost no common carotid artery.

Oertel[20] collected nine cases in the literature, of abnormal union of the internal carotid with the vertebral artery. Among these were those of Quain, Lungel, Duret, Lareniecki, Hochstetter, Flesch, Decker, et al.

Comparative anatomy yields some light in regard to the possibility of atavistic sources for this anomaly.

Chaveau[9] stated that there is no internal carotid artery in sheep. According to this author the seventh branch of the external carotid originates from the arteries of the rete mirabile which, posteriorly, is in communication with the spheno-spinous artery. Towards its middle part the twigs gather into a single trunk analogous to the intracranial portion of the internal carotid.

Strangeway,[29] in describing the carotids of *Ruminantia* observed that they arise, as in the horse, by a primitive trunk from the brachiocephalic artery; they break up at the head forming the external carotid and a small occipital artery; the internal carotid is wanting, its function being performed by the encephalic artery.

Poynter[22] makes the interesting philosophical observation that it is undoubtedly incorrect to interpret all variations as atavistic. He notes that a good many more variations occur in man than in lower animals and that this instability may be due to progressive changes toward a type not yet established but which will more clearly meet the requirements of the organism. He states, however, that if the anomaly is found below man it is retrogressive. (This con-

clusion might be questioned on the basis that evolutionary processes have frequently adopted devious routes to accomplish the same ends in various species of animals.)

Bremer,[5, 6] by his experiments on chick embryos, reveals how relatively easy it is to force the vascular system to alter its wonted course. He states that, "A tortuous course is doomed to obliteration at its weakest part when in competition with other more favorably placed channels." He found an intimate relationship between the positional development of certain nerves and that of the coeliac artery in the chick.

Congden and Wang[10] state that, "In evolution of the vascular system the vessels are exposed to remodelling and shifting of the structures around them and to the blood stream within."

Altman[1] observed a bilateral loss of the middle meningeal arteries in a case of malformation of the external and middle ear. Microtia, congenital atresia, absence of the ossicles, and ossicular muscles, all existed in his case. There was no foramen spinosum. Altman's observations, like those in the present article, were made from serial sections of the petrosae.

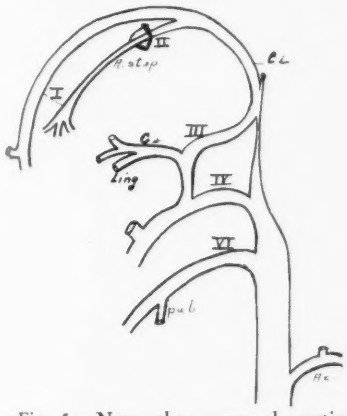

Fig. 5.—Note the narrowed aortic stem between arches III and IV. This may have been the location of the atrophy in the case described. (After Tandler)

DISCUSSION

The internal carotid, as embryologists have long since demonstrated, has a complicated origin from three sources. These are (1) a root portion derived from the third aortic arch, (2) an intermediate portion derived from the dorsal aorta between the third and first aortic arches, and (3) a distal portion which is a continuation of the dorsal end of the first arch towards the developing brain. These stages of development are well illustrated by Tandler.[33]

An explanation of the anomaly here presented is difficult, especially in the light of the statement in the prosector's protocol that the neck vessels were normal in origin and distribution. One can only surmise that there must have been some connection between

the vertebral artery and the internal carotid within the skull and beneath the dura, such as those described by Quain, Smith, and Shepherd. The potentialities for such a condition are illustrated in Fig. 5 taken from Tandler's illustration of the rat embryo, which according to him is quite comparable to the human.

The importance of the internal carotid artery and its tortuous course through the petrosa of the temporal bone are not fully appreciated. This vessel is normally the main source of supply for the eye and a large portion of the brain. Its terminal branches are the ophthalmic artery, the middle cerebral and the anterior cerebral arteries. In its passage through the petrosa the vessel gives off only a small branch toward the eustachian tube. The internal carotid makes two right angled turns between its cervical portion and its intracranial portion. Its bony channel in the petrosa is normally much larger than the caliber of the vessel but cases are on record, as seen in sections of the petrosa, where the vessel practically fills its bony canal. The vessel is surrounded by a pericarotid venous plexus which drains from the cavernous sinus to the jugular bulb. It is also surrounded by a carotico-sympathetic nerve plexus, composed of two main nerve trunks travelling on each side of the vessel wall and anastomosing at frequent intervals. The muscular wall of the vessel is easily recognized as that of an artery as seen in the sections of normal specimens. The lumen is normally widely patent, circular in cross section and heavily coated with muscular tissue. D'Avino[12] found that the amount of elastic connective tissue in the media was markedly reduced as the artery passed through the petrosa. He noted that in the newborn the artery is firmly adherent to the canal wall.

Pathological changes of the internal carotid are frequently seen in routine study of temporal bone sections. These may include atheroma, arteriosclerosis and erosion of the wall. Richards[31] stated: "The carotid is more frequently exposed through erosion of its canal in chronic suppurative middle ear disease than is generally supposed."

To the author's knowledge, atrophy of the internal carotid artery has not hitherto been observed in microscopic sections of the temporal bone. This is the second case on record of bilateral atrophy of the internal carotid.

The author is indebted to Dr. M. Moldovan for the translation from the Italian of D'Avino's article.

Grateful acknowledgment is made to Dr. Benjamin Castleman for making possible the procurement of this material; to Drs. Wislocki, Bremer and Lewis for suggestions regarding the bibliographic significance of the case.

243 CHARLES STREET.

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LXI

PURIFIED GELATIN SOLUTION AS A BLOOD PLASMA SUBSTITUTE

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KALAMAZOO, MICH.

The importance of making available a macromolecular substance which in aqueous solution would exhibit physicochemical properties similar to the blood proteins was recognized during the first World War. It was early appreciated that in addition to possessing these physicochemical properties that substance should be nonantigenic, should be readily eliminated or metabolized and should not cause injury to the liver, kidney, circulatory or respiratory systems and should not show any deleterious influence on plasma protein regeneration or on the factors involved in the clotting mechanism. During the period between the two world conflicts interest in this field of investigation was staggered but was revived in 1939 by the onset of the second World War and also by advancements made in the preparation and clinical uses of human blood plasma and serum albumin. The results of the cooperative efforts of laboratory and clinical investigators indicate that gelatin obtained from bone and specifically prepared for intravenous use under rigid physicochemical and biologic control is a satisfactory blood plasma substitute.

Gelatin solutions were first injected intravenously by Dastre and Floresco[1] for purposes of increasing the coagulability of the blood in cases of hemorrhage. There followed several papers on the use of one to five per cent gelatin intravenously and also subcutaneously in the control of hemorrhage and in the treatment of aneurysm. For this latter purpose gelatin was injected near the aneurysm in the hope that the assumed styptic action of the gelatin would cause a thrombus to form and fill the aneurysm, and later through the organization of the clot prevent rupture and hemorrhage. The parenteral administration of gelatin for these purposes was soon abandoned because of the inability to secure pure gelatin that was free from tetanus spores and also because of the inability to prevent decomposition of the gelatin during sterilization. The action of gelatin in increasing the coagulability of the blood was suggested as being due to

From the Medical Division, The Upjohn Company, Kalamazoo, Michigan.

its calcium content. However, it has never been proved that gelatin has any effect whatever in decreasing the clotting time of blood.

The subject of intravenous gelatin was revived in 1915 by Hogan,[2] who in attempting to secure a colloidal solution as a blood substitute selected a 2.5 per cent gelatin solution. He observed that in cases of shock saline solutions escaped through the blood vessel walls and, therefore, were useless in controlling blood volume and thus combatting the signs and symptoms of circulatory failure. He prepared his solution by mixing 25 gm. of the "purest" gelatin with 1.5 gm. of sodium chloride, adding 100 cc. of water, boiling, filtering through hot filter and autoclaving at 124° C. for one hour. This stock solution was kept in an icebox and was prepared for use by warming and adding it to 1,000 cc. of 0.9 per cent sodium chloride to which was added 2 gm. of sodium carbonate. Six patients with severe collapse due to various causes showed good recovery after intravenous injection of 500 cc. of this solution. Three patients that died showed a temporary rise in blood pressure after 800 cc. Death in these cases was no doubt due to overwhelming infections—typhoid ulcer rupture, hemolytic streptococcic septicemia, and ruptured appendix.

Several years later Bayliss[3] introduced six per cent acacia for treating traumatic shock. He abandoned gelatin solutions because of the tetanus spores reputed to be present in the gelatin and because of the report by Dale and Richards that gelatin given intravenously caused intravascular clotting.

In 1929 Wolfson and Teller[4] reported on the beneficial effects of five per cent gelatin given intravenously in elevating and maintaining the blood pressure in exsanguinated rabbits. Their stock solution was prepared by adding 50 gm. of "Difco" gelatin, 1 gm. of sodium carbonate and 0.9 gm. of sodium chloride to 400 cc. distilled water, boiling for 15 minutes, filtering through glass wool and autoclaving for one hour at 252.2° F. Immediately before use the gelatin was warmed, added to 600 cc. of sterile water and heated to body temperature. Toxicity tests, consisting of injecting 10 cc. of the stock solution into veins of rabbits daily for six days, showed no evidence of intravascular clotting or other ill effects. The animals were used for other experiments and the organs examined postmortem were found to be normal. These observers conclude that:

"Intravenous injection of 5 per cent gelatin solution into animals with low blood pressure due to hemorrhage restores the blood pressure for several hours. The continued presence in the circulation of the gelatin solution is indicated by the low figure reached and maintained by the refractive index. Solutions of weaker concentration are not as effective."

In experimental studies of blood substitutes Waters[5] observed that seven per cent gelatin in isotonic saline injected intravenously in exsanguinated dogs and rabbits in amounts equal to the blood withdrawn was capable of restoring blood pressure to normal. Gelatin was found to exert a favorable influence on maintaining blood pressure and preventing shock for significant periods, probably for 24 hours, at which time appreciable amounts of the gelatin are present in the blood stream.

Gordon, Hoge and Lawson[6] subjected etherized dogs to massive hemorrhage and replaced the volume of blood withdrawn with defibrinated blood, with gelatin eight to ten per cent in isotonic saline or with five per cent glucose in isotonic saline. Their results showed that gelatin was capable of restoring blood pressure usually within the first hour after injection. In this respect gelatin appeared to occupy an intermediate position between blood and crystalloidal solutions. No toxic effects suggesting the presence of toxic proteoses or amines were observed in normal dogs receiving up to 20 cc. of gelatin solution per Kg. of body weight or in dogs after hemorrhage receiving 90 cc. of gelatin solution per Kg. of body weight. Anaphylactoid reactions were not observed in dogs injected with 40 cc. per Kg. of body weight of gelatin solutions nine to eleven weeks after a sensitizing dose of 40 cc. of gelatin solution per Kg. of body weight. Pathological changes were not observed at necropsy in any of the animals that received gelatin.

Parkins, Koop, Riegel, Vars, and Lockwood[7] studied: (1) the response of normal dogs to moderate and large, single and repeated, injections of bone collagen gelatin, with particular reference to toxic effects; (2) the rate and avenue of excretion of gelatin; and (3) the efficacy of gelatin as compared with saline solution and plasma in effecting the return of plasma volume and colloid osmotic pressure of the blood of dogs subjected to hemorrhage or burns of such severity as to be fatal to untreated animals. The general systemic reactions observed, consisting mainly of nausea and vomiting within two or three hours of the injection of gelatin, were similar to those that follow infusion of comparable volumes of saline solution or plasma. An increase in the sedimentation rate of the blood was the only notable hematologic change observed. Kidney and liver function tests and histologic examination of these structures showed no evidence of damage or of gelatin storage. In the normal dog and in dogs subjected to hemorrhage approximately 50 per cent or less of the gelatin was excreted in the urine. The colloid osmotic pressure and the

blood volume in dogs subjected to hemorrhage and burns were noted to be increased as effectively with gelatin infusion as with blood plasma.

Concerning the antigenicity of gelatin it has long been established that a typical incomplete protein such as gelatin is devoid of antigenic power and therefore offers no difficulty from the standpoint of sensitivity.[8-10, 16] Ivy, Greengard, Stein, Grodins and Dutton[11] state that the antigenicity of gelatin is negligible and under present methods of preparation the possibility of bacterial contamination is so exceedingly remote that it cannot be considered to be a reasonable objection. Parkins et al[7] found no evidence of anaphylaxis or sensitization in their dogs that received intravenous gelatin infusions in amounts of 10 to 60 cc. per Kg. of body weight at intervals of days, weeks and months.

Gelatin suitable for intravenous administration must be prepared from appropriate source material under rigid physicochemical and biologic control. The gelatin solution prepared in The Upjohn Research Laboratories is derived from beefbone collagen which is converted to the proper physicochemical state by electrodialysis and by heat under pressure, clarified and rendered free of pyrogens. The final solution made up to five or six per cent in isotonic solution of sodium chloride is autoclaved under fifteen pounds' pressure for that period of time required to produce the desired molecular size and oncotic pressure. The final solution has a pH value of 7.2 to 7.4.

Gelatin and plasma protein molecules differ in size and in shape. The molecules of plasma protein are symmetrical in shape and have diameters approximating 36 angstrom units, whereas the gelatin consisting of linear polypeptide chains has a very long molecule with a diameter of about 18 angstrom units. Beef-bone gelatin has longer molecules than gelatin obtained from soft tissue. With autoclaving, the polypeptide linkages are broken giving rise to smaller molecules, so that the number of smaller molecules increases with the length of time the gelatin solution is in the autoclave. The phenomenon of splitting large molecules into smaller molecules with corresponding decrease in molecular weight is known as degradation. The degree of degradation of gelatin can be controlled by employing a standard technique so that the resulting solution will yield molecules of approximately the same size and the same general molecular weight.

Toxicity studies conducted by Vander Brook and Cartland of The Upjohn Research Laboratories indicate that gelatin solutions specifically prepared for intravenous use are nontoxic. Injected

intravenously in rats in doses ranging upward to 50 cc. per Kg. of body weight and equivalent to 3,500 cc. for man, this gelatin solution was found to cause no ill effects. Rabbits bled by cardiac puncture and injected with an equal volume of gelatin solution twice weekly for four weeks satisfactorily maintained their weight and showed no other deleterious effect of this procedure than a reduction in hemoglobin due to the repeated blood withdrawals.

In a series of dogs subjected to 12 weekly withdrawals of one-third to one-half of their estimated total blood volume and replaced with gelatin equal to 80 per cent of the blood removed, the following determinations were made: concentration of gelatin in plasma and urine, plasma protein nitrogen, blood volume, red cell volume, hemoglobin, red blood and white blood cell counts, sedimentation rate and rectal temperature. Analyses of plasma and urine showed that one-half of the gelatin administered remained in the blood 4 hours after injection and one-fourth after 16 hours. Examination of the urine showed that gelatin was excreted as such but that only 37 to 62 per cent of the gelatin was eliminated. The white blood cell counts showed no significant deviation from the original. The red blood cell counts and the hemoglobin reduced by each bleeding approximated the original figures at the end of the experimental period. Regeneration of hemoglobin therefore was not disturbed. Plasma protein nitrogen determinations revealed no interference with the regeneration of plasma proteins. Total blood volume appeared to be effectively restored by the gelatin infusions. The only findings that showed any real deviation from normal that could be directly attributed to the gelatin infusion was the change in sedimentation rate. The sedimentation rate was greatly increased and was found to be roughly proportional to the plasma gelatin concentration. Return of original sedimentation rate values occurred less than one week following infusion.

Microscopic examination of the heart, arteries, veins, lung, stomach, gall bladder, intestines, adrenal gland, spleen, and muscle and nerve tissue showed no pathologic change traceable to gelatin infusions. Certain gelatin infusion solutions that contained a preservative produced in some instances thrombosis of the infused vein following one injection. With gelatin solutions containing no preservative such local thromboses did not follow single injections. However, upon repeated injections into the same vein gelatin solutions without preservative did cause thrombosis. To eliminate the chance development of thrombosis, therefore, it is advisable to avoid employing the same vein for administering multiple injections of gelatin.

Examination of liver sections showed an increase in sudan staining fat-like droplets in the Kupffer cells. Because the Kupffer cells of dogs that received no gelatin injection were free of these droplets and because the droplets appeared immediately following the gelatin injection and decreased a day of more later, the injection appeared to be directly implicated. In view of the fact that the Kupffer cells showed no nuclear changes and also that other parts of the reticulo-endothelial system showed no change, the presence of the fat droplets in these cells was believed to be a part of a probable metabolic process which did not impair the cellular functions.

Histologic examination of the kidney of gelatin-injected dogs showed that epithelial cells of the proximal convoluted tubules were dilated and occasional cells were vacuolated, presenting a picture which has been described as a protoplasmic disturbance which was probably produced by the excretion of gelatin. In no instance was frank tissue damage such as necrosis observed.

The effectiveness of gelatin solution in preventing experimentally induced shock in dogs was determined by Swingle's group.[12] These Princeton investigators showed that as far as could be judged by the effect on blood pressure, hematocrit and hemoglobin changes gelatin was approximately as effective as plasma in preventing trauma. It was observed that intermittent gelatin infusions of 6.6 per Kg. of body weight for a period of eight hours was effective in maintaining blood pressure and preventing hemoconcentration in 73 per cent of the traumatized animals. Similar amounts of pooled, heparinized dog plasma administered in a similar manner proved no more effective in preventing shock than gelatin.

The effects of gelatin solutions administered intravenously in human beings not in shock and in shock were studied by Kozoll, Popper, Steigmann and Volk[13] of the Cook County Hospital, Chicago, Illinois, and by Jacobson and Smyth[14] of the Department of Medicine, Wayne University College of Medicine, Detroit, Michigan, and Eloise Hospital, Eloise, Michigan. These and other groups of investigators found that gelatin exerted a satisfactory colloidal osmotic effect as shown by demonstrations of significant increases in plasma volume in all patients not in shock and in a large percentage of patients in shock. Following the administration of 1,000 cc. of gelatin solution in a period of one and one-half or more hours to patients in various types of postoperative, traumatic and hemorrhagic shock, plasma volume and blood pressure were effectively increased for at least 24 hours and the major circulatory defect in this condition was corrected in a large majority of cases. While the colloidal

osmotic properties of gelatin solution gradually subside as the gelatin is eliminated in the urine, one infusion may be sufficient to effect a permanent relief from the state of shock and cause the plasma volume to increase and remain at a high level even after the gelatin content of the plasma has decreased. However, in many instances particularly in those patients in severe shock, two or three infusions of 1,000 cc. each may be required.

The fate of gelatin injected intravenously in man has been studied, but the available data indicate that up to 80 per cent is excreted in 48 hours and the fate of the remaining 20 per cent is unknown. Blood urea and blood amino acid levels were not increased by gelatin administration. These findings may be interpreted as indicating that the retained gelatin is not metabolized and that gelatin infusions cannot serve from a nutritional standpoint as a means to maintain a patient in positive nitrogen balance. It may be that this fraction of injected gelatin is stored or converted into other types of protein. However, proof of this is lacking. Histological examination of tissues from moribund patients who had received gelatin supplied no evidence that storage of gelatin in the liver or other tissues had occurred.

Intravenous administration of gelatin was well tolerated and did not give rise to untoward harmful reactions. In a few instances there occurred slight elevations in temperature. Patients observed for over one month following gelatin infusion presented no evidence of undesirable effects of the injection. The blood sedimentation rate was markedly increased in all cases. However, no untoward symptoms appeared that could be in any way attributable to this phenomenon. This increase in sedimentation rate persisted for periods varying from one to five days and was not altered by subsequent infusions of saline solution, dextrose, whole blood or blood plasma. In certain instances it was observed that gelatin injection interfered with blood typing and because of this it is advisable to withdraw a sample of blood for typing from each patient before gelatin is administered.

CONCLUSIONS

As reported by the Subcommittee on Blood Substitutes of the Division of Medical Sciences of the National Research Council,[15] gelatin solutions prepared in specially constructed plants under rigid physicochemical and bacteriologic control were found suitable for intravenous injection without evidence of producing sensitization and without histological or clinical evidence of toxicity or irreversible

accumulation. Such gelatin solutions have a definite effect in increasing the circulating blood volume and cardiac output in dogs subjected to hemorrhage and in patients suffering from hemorrhagic traumatic shock.

Clinical evidence has been presented to show that gelatin solution exerts a definite but temporary effect in relieving the state of shock and that therefore these solutions have a place in medical therapy to restore a loss of circulating blood volume in acute injury of various types and that such gelatin solutions do not contribute significantly to nutrition.

While it is recognized that human whole blood plasma or serum albumin when available are solutions of choice for the treatment of hemorrhage or shock, properly prepared, pyrogen-free gelatin solution makes a satisfactory blood plasma substitute worthy of further clinical trial.

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LXII

TREATMENT OF SINUSITIS IN CHILDREN

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To Dr. Dean we owe a debt of gratitude for his influence in reactivating the interest of the otolaryngologist and the pediatrician in the significance of sinus infection in infants and young children, when in 1918[1] at the meeting of the American Laryngological Association, he presented his comprehensive thesis on "Infections of the Paranasal Sinuses in Infants and Young Children, with Special Reference to Adenoids and Chronic Tonsillitis as Etiologic Factors."

He cited the works of Coffin, Wood, Coakley, Mosher, Meyer, Skillern and Sluder as pioneers in this phase of otolaryngology, in stressing the frequency of its occurrence as contrasted with the infrequency of its recognition.

While in his earlier opinions he mentioned inadequate and injurious dietary habits as factors predisposing to sinus infections, he later became more specific in placing the blame on the allergic diathesis of the individual. His attitude towards treatment was always conservative, reserving surgical measures for the cases with more serious constitutional prognoses. The opinion of the pediatrician was always given serious consideration before surgery was advised.

The incidence of sinusitis in children cannot be accurately stated in percentages because of a variance due to many inconstant factors, the most common of which are the environmental influences to which the child is subjected, the manner of living, the housing, the care by the parent or guardian of the well or sick child, injudicious clothing and improper feeding. It is axiomatic that the more unfavorable the above factors are, the greater will be the incidence of the sinusitis following the common cold, and of the recurrences of the sinusitis, until a chronic condition is established. However, many such unfavorable factors may pertain in families in which children should have much better care. Thus, we are faced with the same problem in all walks of life. It, therefore, becomes the responsibility of the

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physician, in lieu of the parents who fail in theirs, to see that the child is properly cared for in the treatment of his sinus infections.

The incidence is greater in the more inclement weather of the winter months, especially in the latter months when the virulence of the infecting organisms has been enhanced by human passage, and the resistance of the child is at a lower level, whereas in the summer months the rapidly increasing use of swimming pools is also instrumental in causing an increase in the number of sinus infections.

Sinusitis does not occur at any one age more than at another. The extent of the involvement is modified in younger children in accordance with the time of development of the sinuses. According to Schaeffer,[2] the maxillary sinus can be identified in a three-month-old fetus, and is well established at birth, being approximately 8x6x4 mm. The ethmoidal cells are also present at birth, while the sphenoid exists as a spheno-ethmoidal cell and is nasal in position, and the frontal as a fronto-ethmoidal cell. Neither of these sinuses establish their identities until the third or fourth year, although they may grow to a formidable size before that age. It is well to remember that the anterior and posterior ethmoidal cells are considered such according to the location of their ostia where the anlage of the cell originated, the former being in the middle meatus anterior to the lamella of the mid-turbinate and the latter in the superior meatus, irrespective of the developmental perigrinations of the cell itself.

The ethmoidal and maxillary sinuses are apparently involved in equal proportions, although the latter may be infected from the former because of their more dependent positions. However, by the time the patient passes through the hands of the general practitioner to the otolaryngologist with a well-established infection, it is impossible to decide which group of sinuses were primarily affected. Usually one or the other appears to be the major offender.

In our experience sinusitis occurs more frequently in boys. The most evident reasons we might advance for this disproportion are that boys are more careless in adequate protective dress, in their habits of play and in their regard for minor upper respiratory infections.

Physiology—The fact has been incontrovertibly demonstrated by Proetz[3] and Hilding[4] that the two most important mechanisms in the nose are the coating of mucus and the ciliary activity of the mucous membrane, the former functioning as a protective "blanket" and the latter as the motive power to keep this "blanket" moving toward the ostia of the sinuses and thence to the nasopharynx, carrying its enmeshed bacteria, cells, cell fragments and foreign particles

out of the nasal cavity. They stress the importance of a constantly moist surface and the danger of areas of dryness of the epithelium, which render it vulnerable to penetration by bacteria and the establishment of infection in the subepithelial tissue. This is likely to occur if the flow of mucus from the mixed serous and mucous glands of the subepithelial tissue is interrupted and the surface "blanket" is dried by reason of the absorption of fluid by the inspired air.

The cavernous blood sinuses in the inferior turbinates, and to a lesser extent in the middle turbinates, contrive to keep the tidal air currents properly warmed in order to protect the lower respiratory passages from an irritation which they are ill-equipped to meet.

Olfaction is of much greater importance than is ordinarily realized in our evaluation of the significance of sinus infections in children. Adults have frequently experienced the loss of smell and taste in the course of nasal infections, but have been able to compensate for this loss by a conscious persistence in the maintenance of an adequate nutritional state. Usually this anosmia is short lived and of no constitutional significance. But in a child too young to interpret such a phenomenon, or to adapt itself to it, since no similar mental compensatory mechanism can become established, the ill effects may attain a much greater magnitude. The olfactory areas extend over practically the entire superior turbinates from before backward, the anterior fourth of the middle turbinates, and the opposite areas on the septum. Since these areas are located in the narrowest part of the nasal cavities, it is easy to conceive of their being blocked and the olfactory function obtunded by the least turgescence or edema of the superior and middle turbinates, and to remain so for variable lengths of time.

Pathology—Inflammation of the mucosa of the nose and paranasal sinuses of a child is more profound than that of an adult, because the constituent structure more nearly approaches the embryonal type. The epithelium is more fragile, the vessel walls more delicately constructed and consequently more permeable, the tunica propria less fibrous, and the structural cells of a less mature type. It is therefore more vulnerable to irritation or invasion by bacteria and the reaction more intense, because of the facility with which a transudate or exudate may become established in this type of tissue. Thus the cilia may quickly become inactive by reason of the surface contact of the epithelium of the engorged mucosa in the small sinuses and the nose with the opposing epithelium. As the infection progresses and a sinus cavity returns, the sinuses are unable to extrude the fluid content which follows the partial disengorgement of the mucosa with

the sinus while that at the ostia and in the meatuses may remain engorged and edematous. The safety valve of the maxillary sinus is in many cases the accessory ostia, for it is difficult to conceive of the natural ostium, located as it is in the depth of the narrow infundibulum, affording a pathway for the early spontaneous drainage of secretions.

The clinical pathological types of sinus infections in children are essentially the same as in adults, namely, the intumescent, the catarrhal and the purulent. The microscopical picture consists of an edema of the epithelium and subepithelial tissue. The epithelium may undergo degenerative changes and the cells be cast off singly or in blocks leaving a denuded edematous basement membrane. The subepithelial tissue may be infiltrated with red blood cells and the various defensive cells, the round cell, the plasma cell, the polymorphonuclear leucocyte, the endothelial cell, the histiocyte, the lymphocyte and the young connective tissue cell. The relative numbers of each of these cells may vary according to the stage and the severity of the infection. The larger accumulations of polymorphonuclear leucocytes will in some instances constitute minute abscesses, which if close to the surface may rupture through an intact or partially disintegrated epithelium into the sinus cavity. Individual cells may likewise migrate into the sinus cavity through an intact epithelium. We have demonstrated that the spread of the infection through the subepithelial tissue takes place by direct extension and through the lymph and blood capillaries to other locations within the same tissue or to neighboring tissues or lymph glands. The process of repair entails the removal of the bacteria and disintegrated cells by the phagocytic cells, principally the histiocytes, which by their pseudopodial activity insinuate themselves into the lymphatics and are thus carried away.[5] The degree of permanent change in the subepithelial tissue is determined by the number of connective tissue cells which remain behind to become an integral part of the invaded mucosa. The epithelium and the cilia are regenerated from adjacent intact cells and the restitution of the tissue to normal is established, both histologically and functionally.

The chronic hyperplastic type of sinusitis is unusual in children, although we may encounter a polypoid degeneration of the mucosa in allergic states, especially of the antrum.

Predisposing Causes—A child with poor nasal respiration will invariably suffer from more frequent "colds" and develop an infection of his sinuses more often. There may be a thickened septum, or a

deviated septum with or without a high arched palate as an anatomical hazard with which to contend until he reaches the age when it can be corrected. In most cases there will be present hypertrophied adenoid tissue and infected tonsils. The hypertrophied adenoid will not only act as a barrier to the passage of air through the nose, but also to the passage of secretions from the nose. The adenoid itself may become heavily infected and become a constant source of reinfection for the adjacent sinuses.

Of great importance are the constitutional factors which conspire to create a state of lowered resistance on the part of the child. Malnutrition with its forerunner of improper feeding is so often encountered. The feeding problem is one which primarily belongs in the province of the pediatrician, but there may be contributing elements in an abnormal nasal condition which may not be appreciated by the pediatrician. The gustatory sense may be entirely absent because of poor nasal respiration or a block in the area of the olfactory nerve endings, and until these factors are corrected, it will be impossible to arouse in this type of child an interest in food.

It has been stated[6-8] that certain vitamin deficiencies predispose to changes in the epithelium of the sinuses which are of such a nature that infection easily ensues. While these conclusions may be justified by animal experimentation, they have never been corroborated on humans. Nevertheless, a lack of the proper vitamin balance does contribute towards a lack of general resistance which will predispose to infections.

Symptoms—Nasal Congestion—This may occur as a unilateral or bilateral manifestation in the form of a persistent turgescence of the inferior turbinates, often with a concomitant edema of the lateral aspects of the respective middle turbinates, causing obstruction in the middle meatus. While this is frequently an early sign of incipient sinusitis, it is also a significant sign of a dormant, or subacute maxillary sinusitis, without other local clinical signs.

The effects of allergic states upon the nasal mucosa is well known, and when such are coexistent with infection of the mucosa there occurs a magnification of all the symptoms arising from an acute blocking of the sinuses. Consequently, a child who is allergic is at a distinct disadvantage in so far as the incidence of sinus infection in his particular case is concerned.

Discharge—This may be either mucoid or purulent: the former in the early and subsiding stages and the latter in the intermediate, more actively virulent stage of a sinusitis.

Personality Changes may occur, such as irritability, restlessness, languor, anorexia, vomiting, and an indefinable malaise. These are all manifestations of a toxic state resulting from absorption from the focal infection, local discomfort and disturbed nasal physiology.

Headache—Older children may complain of pains in different localities in the head, but an infant cannot. However, we have every reason to suppose that the infant likewise suffers from headache. This pain phenomenon can arise from a blocked sinus, or a toxic irritation, or an actual inflammation of the branches of the fifth nerve as they pass through the sphenoid sinus immediately underneath the mucosa, or adjacent to the ethmoid cells. The vidian nerve is exposed in the same manner, activating a sphenopalatine ganglion neuritis which Sluder[9] has described. Pain may also result from pressure of the ethmoidal nerves between the engorged mucosa of the turbinates and the septum.

Fever—In the acute state of the sinusitis a rise in temperature is usually present, becoming intermittent in the subacute stage and frequently absent in the chronic stage.

States of Malnutrition, especially when accompanied by a fever of unknown origin warrant a thorough investigation of the sinuses. We have frequently seen children who have resisted all ordinary efforts to correct this disturbing condition respond to the proper care of a previously undiagnosed sinus infection.

Complications—These may be local or general. Local complications are due to a direct extension of the infection into adjacent areas leading to the establishment of a more serious condition in these areas. Direct extension occurs through the peripheral bony plates of the respective sinuses. The ethmoidal cells are usually the more common pathways. The superior walls of these cells are extremely thin or entirely absent in some cases, thus bringing the periosteum of the cell in direct approximation to the dura. This also pertains to the lateral wall of the ethmoidal cell immediately adjacent to the orbit, in which the periosteum of the cell is in contact with that of the orbit. It is thus a simple matter for infection to spread to the meninges and to the orbital cavity, leading to a cellulitis or an abscess in that locality. Evidently, and fortunately, this is not a common anatomical anomaly, otherwise these complications would be more frequently encountered. An osteomyelitis of these bony walls may lead to the same complications, however.

In a comprehensive discussion of the sequelae of sinusitis in children, Dr. Dean has mentioned the following complications: bron-

chitis, recurrent fever, gastro-intestinal disturbances in infants, pyelitis, arthritis, phlyctenular conjunctivitis, retrobulbar neuritis, orbital cellulitis, meningitis and brain abscess. That bronchiectasis and peripheral bronchiolitis may be traced to an infection of the homolateral maxillary sinus is still a moot question.

Treatment—It is essential to have a clear concept of the predisposing causes, the physiology and the pathology of sinusitis in children in order to understand and interpret the symptoms and to intelligently plan the treatment. Serious secondary and remote effects of sinusitis may often be the first intimation of this focus of infection, and it is but a futile gesture to treat one or the other as separate entities, as it only leads to eventual failure.

The treatment should therefore be planned according to a simultaneous local and constitutional regime. Dr. Dean, in his many discussions of the successful treatment of sinusitis in children, has repeatedly stressed the importance of the close co-operation between the rhinologist and the pediatrician. Children are only too often not given the benefit of a rhinological opinion unless there is present an obvious nasal infection, especially in conditions of prolonged, unresponsive malnutritional states.

We feel very strongly that the local treatment of a sinusitis should be pursued with meticulous attention to detail, not only of the phase of the infection, but also of the type. The opinion has been expressed by Dr. Dean and others that a large percentage of children subjected to repeated attacks of sinusitis are victims of an allergic background from foods, inhalants or bacteria. The local manifestations of this tendency may be classified pathologically as a hyperesthetic rhinitis, which tends to block one or more of the sinuses and establish a favorable medium for the establishment of an acute or chronic sinusitis according to the frequency of the obstruction. The offending allergens should be traced down and eliminated if we are to expect any favorable results in our treatment of the infection.

Purulent Sinusitis—Our local physical examination and x-ray findings will lead to an accurate diagnosis of this condition and when considered with the general and laboratory findings, we should have adequate information to properly evaluate the significance of the infection. The cardinal principles of aeration and drainage of the sinuses, the *sine qua non* for the proper functioning of the nose and sinuses must be our guide.

The office treatment of a child will have to be modified by the age and temperament of the patient. The general rule to be followed should be to apply all of the principles of adult treatment in so far as the child will cooperate. It is beyond the scope of this paper to discuss child psychology, though it is obvious that it is a large factor in the success or failure of our office treatments. The use of weak cocaine solutions are justified in the irritating phase to facilitate the irrigations of the antrum through the ostium, and the use of small tip suction to cleanse the nose. Displacement irrigations of the sinuses[10] also have a definite place, but here again cooperation is essential.

The home treatment is most important if we are to expect satisfactory results, and a great deal will depend upon the mother or the nurse. When a nasal cavity, or the interstices of the meatuses are filled with pus which cannot be evacuated by the patient, it must be removed by irrigations in order to prepare the nose for the medication to relieve the intumescence of the respective areas. We advise the use of irrigations only in such instances and according to the following technique: an irrigating can or rubber bag containing one pint of warm physiologic sodium chloride solution; an eye dropper or an infant enema tip (a blunt closed tip should never be used) should be held at such a height that it ejects a stream with no more than a four to five inch trajectory. The tip is placed just within the vestibule of the nose. With the patient holding his face on a parallel plane with the floor, or in the case of infants or very young children, lying on a table, on the stomach, an equal amount of the solution is allowed to flow into each side of the nose. The patient is then placed on his back with the head fully extended, a few drops of shrinking solution instilled into each side of the nose and the head kept in this position for at least five minutes, after which the patient is placed on the stomach to allow the medication to infiltrate further into the interstices of the meatuses and to prevent the swallowing of any excess.

The rationale of nasal irrigations is based upon the fact that medication will not reach a membrane or a meatus which is covered with pus or tenacious mucus, and furthermore, the cilia are unable to move such a secretion. By cleansing the surfaces we can establish a condition which will permit both of these elements to function more easily. Irrigations are advised and continued while the patient is under observation, and only as long as warranted by the character of the discharge. This principle likewise applies to the home use of intranasal medication. We have found ephedrine hydrochloride 1½% in physiologic sodium chloride solution to be the least irritating and the most effective. When the patient exhibits a constitutional re-

action to this, neosynephrine hydrochloride ¼% is used. Practically all other intranasal medication irrespective of the theoretical pH has proved to be irritating after short usage.

Surgical Treatment—Dr. Dean has stated[11, 12] that 80 per cent of sinus infections are cured by a proper removal of tonsils and adenoids, while the remaining 20 per cent become our more serious problems, with a good prospect of sinus surgery.

Polypi, when large enough to obstruct respiration or sinus drainage in a meatus, should be removed with as little trauma as possible to the permanent nasal structures. This procedure presupposes adequate allergic studies of the patient. A patient with a high arched palate, in which the cephalocaudal area of the nose is reduced, should have orthodontic measures instituted.

A maxillary sinusitis which has not responded to conservative treatment, in which local or constitutional symptoms persist, is a chronic focus of infection and as such should be eradicated. The great majority of such cases respond to a simple intranasal antral window, and rarely is it necessary to resort to a more radical procedure. An acutely infected, blocked maxillary sinus accompanied by a hemorrhagic nephritis should be opened and drainage established as soon as possible if the kidneys are to be preserved. An antrum puncture in an infant or small child becomes a minor surgical procedure, because of the necessity of administering a general anesthetic.

Extensive sinus surgery in children is unwarranted except in cases of dire emergency. There are instances when a complete and permanent loss of sight is imminent as a result of an optic neuritis with a coexistent sphenoid or ethmoid infection, all other factors having been eliminated, in which an exenteration of these sinuses is justified. An orbital abscess of ethmoidal origin is likewise an indication for surgical interference, the route of approach varying according to the individual case.

Constitutional Treatment—Primarily the general care of a child is in the province of the pediatrician. Nevertheless, Dr. Dean's dictum that an otolaryngologist should be a general practitioner with special training in otolaryngology is sound advice.

While the child is under our care we should make every effort to correct those factors which predispose to infection, such as improper clothing, home care and hygienic surroundings. Insufficient or inadequate diet should be rectified and vitamin deficiencies ad-

justed. Certain anemic conditions may be overlooked unless blood studies are made. Endocrine dyscrasias, especially subthyroid states, have been found to contribute to lowered resistance and a persistent boggy condition of the nasal mucosa, especially in older children. Proper basal metabolic studies should therefore be done and adequate thyroid therapy prescribed if necessary.

Chemotherapy—Space does not permit of an extensive consideration of the increasing value of chemotherapy in the treatment of bacterial infections. In 1938, we[13] proved that sulfanilamide is carried by the blood stream to the mucosa of the turbinates and paranasal sinuses of rabbits in sufficient amounts to exert its bacteriostatic action upon invading organisms. It is logical to assume that a similar action can be attributed to sulfathiazole, sulfadiazine and penicillin if the blood supply to the part is sufficiently active. This has been corroborated by numerous clinical reports. Chemotherapy therefore has a definite place in the treatment of sinusitis, the choice of drug, the mode of administration and the dosage being guided by the type of infection and the age of the patient. We must not lose sight of the fact, however, that the growth of bacteria is inhibited in tissues only and not in cavities and that appropriate drainage of an empyema of a sinus is a most important adjunct to chemotherapy.

In a word, the care of the patient as a whole should always be considered in the intelligent care of the child with a sinus infection.

SUMMARY

1.—In the treatment of sinusitis in children the constitutional imbalance should be properly adjusted by an adequate diet, removal of offending allergens, and supplementary vitamin therapy.

2.—The environmental predisposing factors should be corrected as well as it is within the jurisdiction of the otolaryngologist to do so.

3.—Therapeutic aids should be used as indicated, viz: calcium, iron and thyroid medication.

4.—Every effort should be made to treat the local infection as intensely as conditions permit by the use of suction, nonirritating intranasal medication and displacement irrigations. Nasal irrigations carefully administered with physiologic sodium chloride solution are beneficial in liquefying and removing the purulent secretions and preparing the nose for carefully instilled intranasal medication.

5.—Certain serious constitutional diseases have their origin in latent as well as manifest sinusitis, which may at times be unsuspected.

6.—The ultimate success of the treatment of sinusitis in children depends upon the cooperation between the otolaryngologist and the pediatrician.

MISSOURI THEATRE BUILDING.

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LXIII

DIAGNOSIS OF MANDIBULAR JOINT NEURALGIA AND ITS PLACE IN GENERAL HEAD PAIN

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Any regularly recurring facial pain associated with movement of the lower jaw allows the possible diagnosis of mandibular joint neuralgia, until such condition is definitely ruled out. The degree of this reflex pain may be mild enough to convince the observer that the patient has a chronic complaint with neurotic background. Test of treatment, by the simplest change in occlusion, has relieved several cases of this type, when none of the usual diagnostic findings were present. Also, the degree of pain may be so agonizing as to give a fairly typical picture of tic douloureux. Such a case[1] due to fracture or separation of bone of the tympanic plate was completely studied on the medical service at Barnes Hospital. Trigeminal neuralgia could not be diagnosed. Diagnosis was proved by roentgen study and by the relief attained from fixing the lower jaw with a strong elastic headband.

Little can be added to the vast knowledge of cranial sensations transmitted via the trigeminus nerve. To evaluate pain effects produced by abnormal movement of the condyle of the mandible, it has seemed important to study these functions. The anatomy of the attachment of the mandible to the skull provides very obvious chances for impingement of condyle on sensory nerve branches. Mesially, it may irritate the chorda tympani nerve, as it passes out of the glenoid fossa through the petro-tympanic fissure. Posteriorly it may impact the auricular branches of the auriculotemporal nerve within the temporomandibular articulation, and with wider, loose movement, the condyle may irritate the auriculotemporal nerve itself. When sufficient destruction of joint structures occurs to allow the eroded face of the condyle to rest against the tubercle of the glenoid fossa, abundant source of reflex pain is produced.

In 1934, a symptom complex[2] of pain and subjective ear symptoms was outlined as confusing the commonly encountered head-

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aches of sinus disease. It occurred in cases of malocclusion and destructive conditions of the mandibular joints and was relieved by proper jaw reposition. It consisted of vertex and occipital pain, otalgia, glossodynia and pain about the nose and eyes. The ear symptoms were a sense of impaired hearing or "stuffy" sensation in the ears, tinnitus and grating noises, dizziness relieved by inflation of eustachian tubes, and different degrees of ear pain. The series of such cases at this date is 500 in number.

Wilson,[3] in 1910, elaborated the current knowledge of the auriculotemporal nerve as the most common seat of ear pain; he demonstrated the abundant distribution of its sensory nerve fibers within the external meatus by nerve section. Sluder,[4] in 1918, described a pain syndrome associated with sphenopalatine ganglion disturbance, similar in distribution to the pain areas described above. Lillie,[5] described three cases of otalgia and mastoidalgia, upon which mastoidectomies had been done without relief, relieved by cocainization of the sphenopalatine ganglion. He referred to the work of Wilson explaining this reference of pain along the auriculotemporal nerve. Lyman,[6] in 1924, further showed the relationship of sphenoiditis and related neuralgic pains in the mastoid in the report of a case relieved by sphenoidectomy. Furlow,[7] in 1942, relieved a case of intractable otalgia by isolating and sectioning the nervus intermedius. The patient was suspected of mandibular joint disturbance because of production of the pain on chewing, but was not relieved by change of occlusion. He classed this as idiopathic geniculate neuralgia.

The etiology of glossodynia and burning pains in the pharyngeal wall, without local lesions, was shown as a result of reflex irritation from the auriculotemporal and chorda-tympanic nerves to the lingual and glossopharyngeal nerves in ten cases[8] selected from the first 100 in this series. The proportionate number of cases has varied up to 23 per cent of the larger group.

As to reflex pain from the mandibular joint, otalgia predominates as is shown in the following figures taken from a summary of 400 cases.[9] Some, or all, types of pain were present in each case, and the frequency of the last three types was approximately even: Otalgia, 152; Orbital, cheek, or parietal pain, 107; Glossodynia, 92; Vertical, occipital, and neck pain, 91.

The distributions represent the usual areas of painful fifth nerve hemicranias. The origin of irritation classifies the symptom complex as a secondary or reflex neuralgia. The frequency of occurrence places it in a large, easily classified group.

Frazier,[10] in 1921, classified cranial neuralgias as follows:

1.) post-herpetic neuralgia; 2.) geniculate ganglion neuralgia (Hunt); 3.) sphenopalatine ganglion neuralgia (Sluder); 4.) glossopharyngeal neuralgia; 5.) major trigeminal neuralgia — tic douloureux; 6.) a large atypical group.

Cushing added the neuralgia of tumor involvement. Vail,[11] included the post-herpetic type within the geniculate ganglion group and added a "great superficial petrosal" group, the result of injury to the nerve from petrositis or intracranial surgery.

The diagnosis of mandibular joint neuralgia depends upon history, gross findings, roentgen study, and the test of treatment. From the histories in this series, the following relevant facts are listed. Some are incidents producing acute trismus and mandibular joint injury and other conditions are present a long time with varying grades of chronic trismus. In the latter, the beginning of symptoms follows no pattern, and there is no fixed rule as to severity. Although the rule is that complete loss of the molar teeth invites destruction of the mandibular joints, the paradox is seen almost every day in a person with no molar teeth at all and no evidence of joint changes.

HISTORY:

- Yawning
- Biting of an apple
- Blow on the chin
- Stretching of jaws under anesthetic
- Ulcerated tooth
- Peritonsillar abscess
- Parotitis
- Psychoneurosis
- Fracture of the jaw
- Furuncle in ear
- Tetany
- Tetanus
- Ill-fitting dental plates
- Impacted unerupted teeth
- Malocclusion
- Habit of removing dentures

GROSS FINDINGS:

- Unilateral or bilateral loss of molar teeth
- Overclosure on malocluding natural teeth, or ill-fitting dental plates
- Uneven movement of the lower jaw on opening and closing
- Crepitus within the mandibular joints
- Tenderness of the mandibular joints to internal palpation
- Presence of trismus all grades

ROENTGENOLOGICAL FINDINGS:

- Density changes in the mandibular joint structures
- Narrowing of spaces between the condyle and glenoid fossa, either uniform or asymmetric

Widening of joint spaces and wide excursion of the condyles in the open position

Fixation of one condyle against the tubercle and normal excursion of the opposite condyle

Erosion of the anterior surface of the condyle and posterior aspect of the articular tubercle

Change in normal contour of glenoid fossa, (deepening) and of the condyle, but with normal bone surfaces (suggestive of abnormal stress)

Fracture of the tympanic plate

Impacted unerupted third molar teeth

TEST OF TREATMENT:

Observation of pain changes after fixation of the lower jaw with elastic headgear

Decrease or increase of pain after slight change in closed position of the condyle and fixation with elastic headgear (using cork discs of accurate thickness or, better, temporary dental appliques)

Removal of abscessed teeth, impacted teeth, and correction of all sources of trismus

Dental reconstruction to correct condylar malpositions

The classification of condylar malpositions with the mandible in the closed position was offered by Pippin,[12] as follows:

"1. Unilateral malposition: Right or left normal, right or left malposed.
"2. Bilateral similar malposition: Right and left similarly malposed.
"3. Bilateral dissimilar malposition: Right and left dissimilarly malposed.

"The malpositions are found to be anterior, posterior, superior, inferior, lateral and the possible combinations of these.

"Summarization of results in treatment of mandibular joint neuralgia as in other neuralgias rests on clinical data supplied by the patient. Changes in sensory areas are not recorded except by the patient's report of reduction in painful areas. Relaxation of trismus, when present, is obvious, is usually slow because of necessary readjustment of soft tissue structures, and naturally removes the chief reason for production of pains."

It is difficult to record results of follow-up in this large series which extends over a period of years, because the work was done by many dentists. However, in his series of 100 cases treated under careful supervision, and noting change in condyle positions by serial laminagraphic studies, Pippin[12] reported that "Each of these 100 cases showed definite improvement when treated for correction of condylar malpositions, and 90 per cent, including three diagnosed as having trigeminal neuralgia, were completely relieved of their symptoms."

COMMENT

Mandibular joint neuralgia may be considered of common occurrence and should be investigated in any case of facial pain or headache not accountable from other sources.

The symptom complex embraces some or all of the usual distributions of fifth nerve pain. It belongs in the reflex, or secondary group of neuralgias, in which mechanical irritation of branches reflects pain to standard cranial areas.

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LXIV

NASAL SINUS DISEASE IN CHILDREN, ITS DIAGNOSIS AND TREATMENT

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In the study of 495 children in whom death was due to a variety of causes, Ebbs[26] found nasal sinus disease at autopsy in 30.6 per cent of those under 14 years of age. In infants the percentage was 32.6. Of the 152 cases of sinus infection the maxillary antrum was most frequently involved; that is, in 29.15 per cent of the cases; ethmoids in 13.75 per cent; sphenoids in 15.4 per cent; and in 56.4 per cent of the cases the middle ear was infected. A combination of otitis media and sinusitis was found in 78.2 per cent of the cases involved. Brown[41] quoting Mitchell stated that in the study of 145 cases of sinusitis the ethmoid was involved in all cases, the antrums in 84 and the sphenoids and frontals 2 each.

Regardless of the prevalence of nasal sinus disease it is constantly being overlooked. Most often the attention of the physician is directed to the tonsils and adenoids and the chest. Many physicians are still not aware of the anatomical peculiarities of children's sinuses. Our attention is called[26, 8] to the great individual variations in their size. In the newborn the nasal sinuses are represented only by mucosal folds and recesses. In the first year of childhood the nasal mucosa is thick, rich in blood vessels, mucous glands and lymphoid tissue. It is, therefore, very susceptible to any infection.

The maxillary antrum is present at birth and is little larger than a large pea. It is well developed at about seven years of age. Development of the antrum depends upon the normal dentition.[5] The ethmoid sinuses have reached considerable development at two years and are fully developed at from 12 to 15 years of age. Schaeffer states that the sphenoid sinus is more precocious than one would infer from statements in the literature and that by the third year it has assumed proportions sufficiently large to become the seat of pathologic processes and to retain infectious material in its cavity. Development of

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the frontal sinuses begins in the first year and reaches its full growth at from 12 to 15 years. The frontal sinuses are rarely of clinical significance before the eighth or ninth year.

Infection of the nasal sinuses during their period of development has a very definite influence on their normal growth. Shea[2] reported a series of cases of arrested development of the frontal sinuses which was due to infection in the antrums and ethmoids. A normal development of the frontal sinuses resulted after the other sinuses had been successfully treated. Levy[7] says that in the first two to three years of life there is no real sinus and that any so-called sinus infection is in reality an osteomyelitis. He says that any sinusitis in infancy is always a maxillary sinusitis. I believe, however, that we all have seen orbital complications of ethmoid infection in early infancy. Crooks[4] says that sinus disease in childhood is as frequent as in later life, and that such infection may act as a focus in arthritis, nephritis and gastroenteritis. Dean made this same observation in 1922 when he discussed the complications of sinus disease in children. Brown[15] examined 100 rheumatic children and found sinus infection in all of them. The sinuses are foci of infection in a large number of conditions: asthma, chronic bronchitis, bronchiectasis, and otitis media.[1, 4, 36 38]

Crooks[12] and others have tried to list the symptoms on a percentage basis and nasal discharge, repeated colds, mouth breathing, cough and cervical adenitis are the most common complaints. Other prominent symptoms are: an unexplained daily rise in temperature; poor appetite; inability to gain weight; headache; hoarseness; otitis media; conjunctivitis; keratitis; blepharitis and orbital cellulitis. We are frequently requested to search for foci of infection in such general diseases as: nephritis; pyelitis; chorea; arthritis and rheumatic fever;[10] these are often found in chronic sinus disease.

Van Alyea[3] reported three cases of neglected or overlooked sinus infections in childhood. He states that a persistent sinus disease in children is seldom diagnosed unless orbital cellulitis or some other complication develops. Most often it is regarded as a long-standing cold. Neglect of sinusitis in children is regrettable because with wide ostia these cases can be more easily treated than in adults. A careful history is an important detail. In my experience allergy is very important and in a great majority of cases the soil has been prepared for the secondary infection, resulting in sinusitis. To treat a sinus infection and pay no attention to the allergy would be a waste of time. Inquiry as to the possibility of foreign bodies in the nose should not be omitted.

In making an examination of the sinuses in children our first step is to gain the confidence of the child. I believe this can best be done in the absence of the parents. As a rule there should be little or no difficulty in doing an anterior rhinoscopy. Some solution to shrink the nasal mucous membrane can be sprayed into the nostril and allowed to produce the desired results. I use a solution of cocaine, .25%, followed by neosynephrine, .25%. Many children will not object to one's placing tampons saturated in the latter solution in the nasal cavities. I have found few small children in whom I can make a satisfactory postnasal mirror examination. An application of 2 per cent pontocain to the nasal mucous membrane will, in the average child, enable us to make this examination with the Holmes nasopharyngoscope. General anesthetic may be necessary for this procedure.

Crooks[4] feels that transillumination is of little value but I have found that if it is negative or if the two sides do not transilluminate equally well it is of distinct value. Intranasal examination is not sufficient in certain types of sinusitis. There may be no local signs of sinusitis and in these radiography may reveal pathology.[7] Mitchell[10] insists that a complete history and a general physical examination, including rhinological study, are necessary for an accurate diagnosis of sinus disease in children. Another observer stresses the value of cytology of the nasal secretions, and bacteriological study.[17] Contrast media in radiological examination may prove of value.

Difficulty in diagnosis is recognized by many others.[31,39] Bowen-Davies[27] warns that radiography is not sufficient for a diagnosis of sinus disease in children. In 44 patients with negative x-ray films, antrum puncture revealed infection 9 times. A thickened membrane may be due to allergy and not to a sinus infection. Maresh[30] mentions that a sinus film may show changes which seem pathologic, but they are normal for certain ages. Clinical infection with normal x-ray findings was observed in 42 per cent of all cases, while pathological x-ray findings with corresponding clinical history was found only in 28 per cent.

It has been my custom for years to examine carefully the nasal sinuses of every child who is brought in for advice as to the advisability of having the tonsils and adenoids removed. If there is any doubt in my mind regarding the sinuses I always make an examination with the nasopharyngoscope and do an exploratory puncture of the maxillary antrum at the time the tonsils and adenoids are removed. This latter procedure is carried out in the manner suggested by Dean more than twenty years ago. The instrument consists of a straight cannula

with trochar and a small cannula that replaces the trochar as soon as the antrum is entered. A 20 cc. syringe is fitted to the small cannula and with this we first aspirate the cavity. If fluid is found, it can be removed without contamination and cultured. If no fluid is found, we then inject 10 cc. of sterile water into the antrum and aspirate for the purpose of culturing. With this procedure I have been able to solve many diagnostic problems in suspected infection of children's sinuses.

In the treatment of sinus disease of children one should remember, as Cone[6] says, that children are emotional. However, emotions of children are most easily controlled when the parents are not present. Brown[13] calls attention to the ill effects of prolonged recumbency in sinusitis. He describes many conditions which he feels are due to absorption of toxins from the pent-up secretions in the sinuses. Keeping the head elevated aids in the drainage of infected sinuses.

Mitchell[29] says that treatment should be started only after all etiological factors have been ascertained. He believes that steam-heated homes and too early removal of tonsils predispose to sinus disease. Conservative treatment is recommended by Gewanter,[35] Hall[40] and the majority of authors.

Shea[2] stresses the importance of correcting the allergic disturbance as well as eradicating the infection and in so doing, obtain a permanent cure. Schoenberg[9] mentions that hot air treatment is not tolerated well by infants, and if applied it should be only by short wave diathermy. Kerley[16] recommends tamponage with a solution composed of 2% ichthyol in 10% glycerine. Tremble[21] points out that local treatment should not interfere with the normal physiology of the nose. The pH of solutions used should be between 5.5 and 6.5. A 5% solution of urea or .5% solution of allantoin has been used with good results. Tremble states that only about six per cent of patients will require antrotomy. Local use of ephedrine is recommended by the majority of observers. The local use of sulfonamides in powder, in solution and combined with vasoconstrictors has been attempted with some good results. Irrigation is useful during the acute stage to relieve pain but it should not be used while the temperature is rising.[2]

Gundrum[18] says that the Proetz displacement treatment, using diluted bacterial antigens, may establish a local immunity. He uses a stock vaccine containing staphylococcus, streptococcus and/or colon bacillus. A 10 per cent solution is first used and this is increased until the pure antigen is used. In his series of 32 patients 86 per cent

were definitely improved after 24 treatments. From my own experience I add a word of caution regarding the use of antigen by this method. I have had a few quite unusual reactions from its use. Asherson[25] describes a method of irrigation with four to six ounces of sodium chloride solution containing one-half grain of ephedrine to a half pint. The head is held in the Proetz position, the solution is dropped into one nostril and removed from the opposite side by suction. Four to six ounces is used on each side and the treatment is repeated twice a week for six weeks.

Results from the use of roentgen therapy have not been encouraging. Shea[2] and Paterson[20] have had good results from the use of short wave. The sinuses should be completely drained before the treatment, which lasts 15 minutes and is repeated 10 or 12 times. Negative pressure, used by the Haskin method, the Coffin or the Proetz method, is a recommended procedure. In my hands the most satisfactory vasoconstrictor for use in the nose is .25% neosynephrine hydrochloride, using Ringer's solution with a pH of about 6. to dilute the 1% stock solution.

Dean and his co-workers at Iowa many years ago called our attention to the importance of diet in the treatment of sinus disease in children. He demonstrated sinus infection in animals fed on diets lacking or low in vitamin A. Diet alone will not cure a sinus infection; the source of the infection must be removed and then the proper dietary regimen instituted. In food allergy I have been disappointed in the results obtained from cutaneous tests and have accomplished more by the elimination diet method. Troup[24] stated that the majority of children suffering with nasal sinus disease have a vitamin deficiency.

All sinus disease is not cured by a climate that is warm and dry. A change in climate will often benefit when all other means have failed but no one climate is suitable to all types of cases. Sinusitis with an excessive amount of secretion does better in a climate like that found in Arizona. When there is not much secretion and the patients are more comfortable on damp days, they will do better in a climate like Florida. The moderate climate of Southern California is best suited to children who have most of their symptoms in the winter months in the northern states. California's best sinus climate is located fifty or more miles in from the coast line. In my judgment the patients most benefited from a change of climate are those that have a definite allergic background. In my experience the use of stock vaccine has been disappointing. Autogenous vaccines have been of benefit in some

cases. I have not had experience with the local use of vaccine by spraying it in the nose.

In a general way we must consider a few important etiological factors when we undertake the conservative treatment of sinus disease in children; allergy, improper diet, poor hygiene, lack of sufficient vitamins, and hypothyroidism.

Many years ago Dean reported that 90 per cent of his patients were cured by the removal of tonsils and adenoids. I am not sure that this statement would go unchallenged today. Mitchell[29] said that early removal of tonsils predisposed to sinus disease; in my experience this has not been true. In the presence of allergy with secondary infection of the sinuses, I believe that we will be disappointed in the results from the removal of tonsils and adenoids.

Radical sinus surgery on children is, in my judgment, rarely necessary. McArthur and Harper[3] felt that the Caldwell-Luc operation was the surest and safest way to remove the diseased focus. I believe that the danger of possible injury to unerupted teeth is too great to follow this procedure except on rare occasions. For many years it has been my routine, when nonsurgical measures did not give the desired results, to make a small opening through the inferior meatus with a short rasp. In the cooperative child this will usually permit a sufficient number of irrigations to effect a cure. In the uncooperative child I have had a great deal of satisfaction from the method suggested by Shea:[23] a small self-retaining catheter placed in the rasp opening with the outer end tucked up into the upper part of the nasal vestibule. This permits painless irrigation of the sinus for as long as is necessary. I have left the catheter in place as long as ten days or two weeks. Some authors recommend making the window through the middle meatus. In children I believe that we should use the lower meatus because we will then interfere to the least possible degree with the normal physiology of the nose. Nature usually closes the opening in the inferior meatus and the ostia is left in its original condition. Accessory ostia made by perforating the membranous portion of the middle meatus are, in my judgment, conducive to more frequent antrum infections in adult life. In making the rasp opening through the inferior meatus we should use caution not to come too far forward or too high so that we will be more certain to avoid injury to the lower end of the lachrymal duct.

Lillie states that 98 per cent of the cases of nasal sinus disease in children are cured by conservative treatment.

The treatment of this condition requires much painstaking detail. Our results will depend on the amount of cooperation we get from the child, the family and the family doctor.

Surgical treatment should be instituted only after conservative measures have failed to give results.

1515 STATE STREET.

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LXV

ADENOCARCINOMA OF THE TRACHEA

A PATHOLOGICAL CLASSIFICATION OF ASSISTANCE IN TREATMENT AND PROGNISIS

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Along with an increase in the incidence of carcinoma of the bronchus one finds, in reviewing the literature, a similar increase in reported cases of carcinoma of the trachea. Whether this increase in reported cases is due to a more widespread knowledge and use of bronchoscopy, or whether the increase in cases actually exists, is argumentative. There is a corresponding increase in the number of patients with carcinoma of the trachea in whom cures have been effected for a disease which formerly was considered invariably fatal.

Carcinoma of the trachea is a relatively rare lesion. Fraenkel,[8] in 5,063 autopsies, found 7 cases. The series from the Mayo Clinic[7, 14] in 1939 totalled 16, 9 of which were adenocarcinoma. D'Aunoy and Zoeller[5] found but one case from the records of the Charity Hospital, New Orleans. Culp[4] was able to find but one case in 9,000 autopsies at the Pathological Institute of McGill University and one in 12,700 autopsies at the Montreal General Hospital. Both of these were squamous cell carcinoma.

In order to treat carcinoma of the trachea effectively it is necessary to have a thorough understanding of the pathology of this condition. Malignant tumors of this organ fall into two main groups, squamous cell carcinoma and adenocarcinoma. Of the latter group there are numerous subclassifications, some of the tumors bordering on the edge of being benign tumors, if not actually being benign.

We are indebted to Krompecher[12] for the modern concept of tumors arising from respiratory epithelium. The basal layer of the pseudostratified epithelium is an undifferentiated structure which has the potentiality of producing squamous, glandular or columnar epithelium. Tumors of the trachea, therefore, may occur in a vari-

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ety of cellular forms as a result of abnormal growth of the basal cells. It is accepted, also, that tumors arise from islands of metaplasia as well as from the mucous glands and their ducts.

Culp,[4] in an exhaustive review of the subject, found squamous cell carcinoma and adenocarcinoma to occur in almost equal frequency, although the tumors in many case reports could not be classified exactly due to inadequate pathological study or description. As a rule squamous cell carcinoma tends to metastasize much more readily than adenocarcinoma. Metastases usually involve the esophagus and the regional lymph nodes, while infrequent reports occur of metastases to more distant organs.

Tumors consisting of glandular epithelium of varying degrees of malignancy occur in the trachea.

Mixed Tumor. A true mixed tumor, identical with that found in the parotid gland, may occur in the trachea. Gerlings and Roegholt[9] report a patient who had symptoms of stridor and dyspnea for 14 years before seeking relief. The mixed tumor was situated in the upper one-third of the trachea and arose from the posterior wall. It was removed by opening the upper trachea through an anterior neck incision.

Howarth[11] reports a case of mixed tumor arising from the right lateral wall of the upper one-third of the trachea. He considered it to arise from the mucous glands of the trachea. Excision by thyrofissure was performed twice to remove the entire tumor. One year after treatment no evidence of recurrence could be found.

These tumors are slow growing and should be treated the same as mixed tumors of the salivary glands.

Basal Cell Carcinoma. In the trachea as in other organs lined by pseudostratified columnar epithelium, there may occur a tumor microscopically undistinguishable from basal cell carcinoma of the skin. Great confusion has arisen concerning this tumor because of the multitude of names it has been given, depending on certain minor variations in architecture. Cylindroma, adenoid cystic epithelioma and adenocystic carcinoma are other names for the same tumor.

Ewing[6] uses the term cylindroma in two different ways. In discussing regressive changes in tumors he states,

"Cylindroma, or syphonoma, are terms applied to a tumor structure in which the stroma appears in the form of elongated, twisted, thickened cords of hyaline

material. This structure is most often seen in basal cell carcinoma. In old regressing carcinoma large areas of tissue may show very few tumor cells in a mass of quiescent hyaline stroma."

In his chapter on tumors of the salivary glands he says,

"Under the term cylindroma has been described a rather frequent form of adeno-carcinoma of the salivary glands. These tumors are composed of anastomosing cords or broad masses of epithelial cells inclosing many spaces filled with mucus. There are coarse trabeculae of connective tissue which may undergo hyaline or mucoid degeneration. The structure suggests a relation to basal cell carcinoma of the type of adenoid cystic epithelioma, but Lowenbach was able to trace the origin in one case to the ducts and in another to the acini of the gland."

Basal cell carcinoma, according to Krompecher,[12] arises from the basal cells of the respiratory epithelium which he states are closely related to the basal cells of the skin and hair follicles. One sees cells with medium-sized round basophilic nuclei growing in anastomosing cords. The number of acini varies in different tumors and in different parts of the same tumor. Acini, when present, usually contain mucus giving a ground glass or thready appearance. There is a connective tissue stroma which often undergoes hyaline degeneration.

These tumors often arise from the posterior tracheal wall just below the glottis. Their slowness of growth is evidenced by the fact that all cases reported had cough, stridor or dyspnea for one year or longer before the true nature of the lesion was discovered. A large percentage received treatment for asthma for varying lengths of time.

Cann[3] reported two cases of basal cell carcinoma treated by x-ray therapy alone and in each case the tumor disappeared completely.

One patient with a tumor in the upper one-third of the trachea had symptoms of dyspnea and stridor for one year. The other, with a tumor in the lower one-third of the trachea, had had a hemoptysis four years previous, cough for two years and inspiratory and expiratory stridor for one year.

Boemke and Moritz[2] report a cylindroma of the posterior wall of the trachea in its upper one-third successfully removed by tracheofissure. The operation was followed by x-ray irradiation.

Neilson[13] reports a patient who suffocated due to tumor of the posterior wall of the trachea just above the carina. Symptoms had been noted for 21 months. The microscopic appearance of the tumor was "reminiscent of rodent ulcer."

Tiling's[16] patient dated his symptoms from an attack of grippe 18 months previous to operation. By tracheofissure the author removed a basal cell carcinoma which arose from the left wall of the trachea from the cricoid cartilage to the seventh tracheal ring. Radium was used subsequently.

Baratoux[1] reported one case with a good result from x-ray therapy alone. Increasing dyspnea had been present for one year during which time the patient was treated for asthma. The tumor arose from the right lateral wall of the trachea, 3 cm. below the glottis.

Guttman[10] reported a case with a similar tumor extending from the subglottic region down into the right main bronchus. Dyspnea had been present for two years. Tracheofissure was performed to relieve urgent dyspnea. X-ray therapy was advised later because of the extent of the infiltration. A year later the patient was living although still wearing a tracheotomy tube.

Malignant Adenoma: These tumors, true adenocarcinoma, arise either from the basal epithelial cells, the mucous glands or their ducts. The morphology varies according to the degree of malignancy. Acini may be produced in profusion or the tumor may appear solid for the most part with only occasional glandular structure. They may be graded according to their malignancy and the treatment and prognosis determined to better advantage.

Although patients with mixed tumor or basal cell carcinoma usually give a history of long-standing dyspnea, this may also be the case in patients with adenocarcinoma. As a rule, however, symptoms are of short duration in the more malignant cases. Exceptions appear in a case of adenocarcinoma reported by Simpson and Moore[15] in which dyspnea was present for two years. One patient reported by Baratoux[1] was thought to have a gumma of the trachea until the diagnosis of adenocarcinoma was made two years later.

The largest series of cases of tracheal carcinoma reported comes from the Mayo Clinic.[7, 14] Of the 16 cases, 9 were adenocarcinoma. Three were in the upper one-third of the trachea, the remainder being in the middle or lower one-third. Figi considers those originating in the lower one-half to be invariably fatal. Those in the upper one-half should be treated by cautery excision followed by radium therapy. Those of the lower one-half may be temporarily relieved by the same procedures employed endoscopically. A case of adenocarcinoma reported by Baratoux,[1] however, responded to x-ray irradiation alone.

The following case reports are of adenocarcinoma of the trachea.

REPORT OF CASES

CASE 1.—A 66-year-old white woman was admitted to the hospital on March 4, 1938, with a number of complaints beginning five months previously and increasing in severity. Her illness began with cough, malaise and fever. The cough was productive and the sputum soon became blood-tinged. For several weeks she had chest pain which was diagnosed pleurisy. Dyspnea and frank hemoptysis began more recently. There was a loss of 28 pounds during the five-month period.

For seven years the patient had been working in a plate glass factory where the air was very dusty.

Five years previous to this hospital admission she had had an adenocarcinoma of the corpus uteri for which hysterectomy was performed. It seemed certain that this tumor was entirely within the uterus.

When hospitalized the patient was experiencing mild dyspnea, especially when lying down. There was weakness, fever, leucocytosis and a mild secondary anemia.

Over the right upper lobe there was dullness, tubular breathing and increased whispered voice.

X-ray examination of the chest showed atelectasis of the right upper lobe.

Tubercle bacilli could not be found in the sputum.

The patient was thought to have a carcinoma of the right bronchus and on March 10 a bronchoscopy was performed. In the lower third of the trachea were numerous smooth pedunculated polypoid tumors attached to the tracheal wall so loosely as to be shorn off by the lip of the bronchoscope. Some were coughed out through the bronchoscope and others were removed with forceps. One large tumor hung down into the right main bronchus and obstructed the opening of the right upper lobe bronchus. Bleeding was only moderate.

Microscopic examination (Fig. 1) showed several tumors made up of granulation tissue densely infiltrated with plasma cells and small lymphocytes. The surface of these had patches of stratified

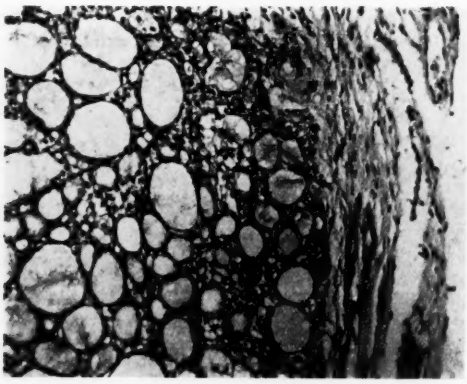

Fig. 1.—(Case 1) Photomicrograph of adenocarcinoma.

squamous epithelium. Throughout were islands of anaplastic columnar cells forming acini. Mitotic figures were numerous. *Microscopic diagnosis*: Adenocarcinoma, grade IV.

X-ray therapy was given and the patient left the hospital. A year later she expired. The postmortem findings disclosed extension of the tumor to the right lung and mediastinal lymph nodes.

CASE 2.—A 40-year-old white male was admitted to the hospital June 30, 1942. A year before he began to notice difficulty in breathing following exertion. Six months before admission he experienced an acute attack of dyspnea preceded by a feeling of heaviness in the chest. He had great expiratory difficulty although inspiration was normal At times he could hear a rattle in his chest. He had no pain. Attacks of dyspnea became more severe and more frequent and were accompanied by blood in the sputum. During the past six months he lost 20 pounds. He had been treated for asthma.

When admitted the temperature was 37.4° F., pulse 84, respiration 20 and blood pressure 150/75. He was exerting much force to empty his lungs. Loud coarse rhonchi were heard over the right lung. There was stridor over the trachea which was more marked when the patient was on his left side.

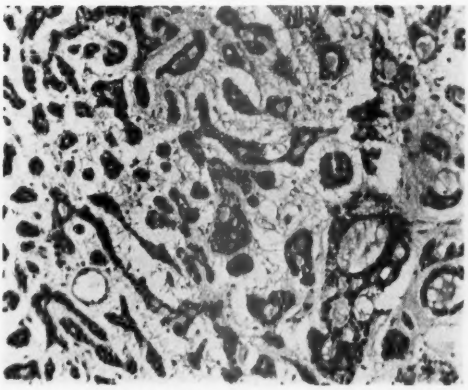

Fig. 2.—(Case 2) Photomicrograph of adenocarcinoma.

Laboratory findings were not significant. X-ray films of the chest including laminagrams showed a soft tissue mass about 3.5 cm. in diameter encroaching upon the trachea just above the carina.

Three days later there was a severe attack of dyspnea and cyanosis that failed to respond to adrenalin, aminophylline or intracardiac coramine and the patient expired.

At autopsy it was found that both lungs were distended, especially the right. Both were congested on the surface and contained some emphysematous blebs but were crepitant throughout. At the junction of the trachea and the right bronchus rose a cauliflower-like mass extending into both the right bronchus and the trachea, filling the trachea to such an extent that not more than a 2 mm. lumen existed. A cut section of the mass showed a gray-white tissue with small areas of hemorrhage and necrosis.

Microscopic examination of the tumor showed it to contain medium-sized cells with clear cytoplasm and basophilic nuclei containing granular chromatin and an occasional nucleolus. The cells were arranged so as to form a double layer about oval or round clear areas which occassionally contained a faintly-eosinophilic material. The regional lymph nodes showed no tumor. *Diagnosis*: Malignant adenoma, or adenocarcinoma, grade III.

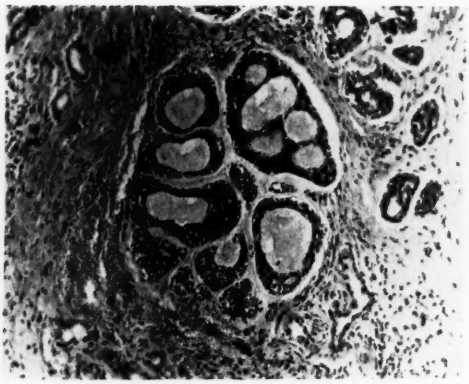

Fig. 3.—(Case 3) Photomicrograph of basal cell carcinoma.

CASE 3.—A 29-year-old white man first noticed difficulty when he had an upper respiratory infection two months before admission to the hospital. Dyspnea soon set in and treatment for asthma was given. On November 3, 1941, he came to the hospital with urgent dyspnea. An attempt to pass a bronchoscope failed due to the presence of a large tumor mass about 2 cm. below the glottis. A tracheotomy was performed as low as possible and the tracheotomy tube, when inserted, could be seen to pass just below a beefy-appearing round smooth mass nearly filling the upper trachea.

On November 22, 1941, the trachea was exposed and opened through the second to the sixth rings. This revealed a rounded mass attached to the posterior tracheal wall from rings two to five. Using a semisharp elevator the mass was dissected away from the tracheal wall and removed. Bleeding was not troublesome. The trachea was closed except for the tracheotomy tube which was removed three days later.

Microscopic sections of the tissue removed at operation consisted of a mass of connective tissue covered with stratified squamous epithelium except where ulcerated and infiltrated with masses of medium-sized cells with scant faint blue cytoplasm and small pyknotic round or oval nuclei. These cells formed acini in many areas and solid masses or cords in others. Mitotic figures were not seen.

Most of the acini contained mucus. Hyaline changes were present in the connective tissue. *Diagnosis*: Basal cell carcinoma.

Roentgen therapy was administered to the upper trachea before the patient was discharged. When seen on May 1, 1943, no evidence of recurrence could be detected.

SUMMARY

Roughly one-half of the malignant tumors of the trachea are glandular in structure. Accurate pathological study is an aid to correct treatment and prognosis.

Mixed tumor and basal cell carcinoma, especially if found in the upper one-half of the trachea, should respond favorably to excision followed by irradiation therapy.

Adenocarcinoma (malignant adenoma) of the lower one-half of the trachea is invariably fatal. In the upper one-half of the trachea cautery excision followed by irradiation is the treatment of choice.

Two cases of fatal adenocarcinoma and one case of basal cell carcinoma with a good prognosis are presented.

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LXVI

OTORHINOGENIC HYDROCEPHALUS

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That a hypertensive intracranial state of inflammatory origin may complicate infections of the ear and paranasal sinuses is well recognized. Williams,[1] in 1938, prophesied that the advent of chemotherapy with increasing cures of septic otitic meningitis would increase the problem of otitic hydrocephalus. More recently Reeves,[2] reporting cases secondary to severe sinusitis, suggested that otorhinogenic hydrocephalus is a more suitable term as it would also include those similar cases following infections of the sinuses. As recently as January 1944 in a summary of bibliographic material concerning otitis media and its complications[3] there is no mention of this hypertensive intracranial complication. It is therefore considered justifiable to call attention once again to the possibility of such a complication of any severe infection in the ear or sinuses by reporting an unusually complicated case.

Beginning with reports of similar and allied conditions in the nineteenth century a rather extensive literature has accumulated. This includes several classifications, a confusion of names, a fairly definite clinical syndrome, and considerable experimental work. However, it is apparent that a few important developments are most responsible for the evolution of our understanding of this condition. In 1891 Quincke[4] was probably the first to treat such a case by lumbar puncture, and he is credited with originating the procedure of lumbar puncture. In 1913 Passot[5] was probably the first to establish the clinical picture, its rational basis, and to suggest a plan of adequate treatment. In 1931 Symonds[6] suggested the term otitic hydrocephalus. By 1935 approximately 60 cases could be collected from the literature, and there have been numerous additions since. After a very extensive review of the literature, in 1938 Williams[1] stated that the term otitic hydrocephalus "while not completely accurate, serves to keep otologists on the alert for its presence with an

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otitis, and is, therefore, a generally useful term." It would seem that the recently suggested otorhinogenic hydrocephalus merits consideration.

Passot's classification, which is probably the best, considered that an aseptic meningeal state of otitic origin causes a hypertensive intracranial state, or a true aseptic meningitis. By way of differentiation he explained that the hypertensive state is an outpouring of cerebrospinal fluid without cellular elements or albumin, while the true aseptic meningitis exists when there is an increase of cellular elements and albumin in the cerebrospinal fluid. He further subdivided the hypertensive state into meningeal hydropsy and an acute internal hydrocephalus. These two usually coexist, but internal ventricular hypertension could occur because of occlusion of one of the foramina of Monro or of the aqueduct of Sylvius. Passot wrote that the true hypertensive state was characterized by enormous excess of cerebrospinal fluid in arachnoid or ventricular cavities, a clear fluid without cellular elements and without albumin, and in which draining of the excess of cerebrospinal fluid produced immediate and definite relief. That an apparent aseptic meningeal reaction may be the preliminary state of a suppurative meningitis, and also that a septic localized or generalized meningitis may cause increased intracranial pressure are well recognized in the literature. Other classifications have been suggested which seem to present no advantages over Passot's.

Different authors have called this intracranial hypertension by numerous names. Among those more commonly used are serous meningitis, meningitis sympathica, ventricular meningitis, acquired hydrocephalus, cerebral pseudotumor, pseudo-brain abscess, and otogenous encephalitis. Probably because of the confusion of this multiplicity of names, Symonds suggested the term otitic hydrocephalus which "implies no active process of inflammation and since it is free from the qualification of internal or external, it includes conditions in which fluid is within both the ventricles and subarachnoid space." The sulfonamide drugs and newer chemotherapeutic agents will save not only otitic patients but also sinusitis patients from developing this type of hydrocephalus. Therefore, the term suggested by Reeves, otorhinogenic hydrocephalus, may be favorably considered.

The causation of the hypertensive state was not well understood until considerable study by numerous investigators clarified the problem. Suggested causes were as varied as the multiplicity of names proposed. However, the weight of evidence now favors the cerebrospinal fluid as being a biological filtrate and not a secretion, and ob-

structive phenomena as the cause of increased intracranial pressure in the hypertensive state. If certain toxic substances are present in the blood or if osmotic tension of the blood becomes low, hydrocephalus may be favored by increased spinal fluid formation. Any pathologic condition which may interrupt the normal route of circulation or absorption of the cerebrospinal fluid may be a cause of otorhinogenic hydrocephalus. Edema of the ependyma and underlying brain tissue has been found in human or experimental subjects by a number of investigators. Subependymal cellular infiltration and degenerative changes in capillaries have been reported. Shapiro[8] blames arachnoiditis. Others believe that some pre-existing pathologic or anomalous change may have diminished the reserve capacity of the foramina, or of the arachnoid and pacchionian villi, or of the perineural lymphatic space, and that the hydrocephalus may be precipitated by sudden edema or inflammation. Anatomical variations in size of the sigmoid sinuses or jugular foramina have been implicated by Ersner and Myers,[9] and are undoubtedly important in certain cases. Dean[10] believed that extension of the thrombus into deeper intracranial venous spaces from the lateral sinus was an important factor in the development of the hypertensive state in lateral sinus thrombophlebitis. It has since been found that cerebrospinal fluid pressure is directly affected by venous pressure, and that if thrombophlebitis or increased intracranial pressure interferes with the escape of blood via the intracranial sinuses, a vicious circle may be produced with ever increasing hydrocephalus. After his thorough study of all the experimental evidence, Williams concluded that this intracranial hypertension may follow any inflammatory or obstructive lesion of the pacchionian bodies of bacterial or toxic origin.

The signs and symptoms of the true hypertensive intracranial state are relatively few. This condition may develop early in the disease or later; frequently it appears when a patient is considered well on the road to recovery. Papilledema is the most prominent sign. It may exist without other signs, and therefore should be watched for routinely in this group of patients. For many years Dean taught that papilledema of lesser or greater degree should be anticipated in approximately 40 per cent of patients operated on for lateral sinus thrombophlebitis. He stressed the early recognition of papilledema to insure the proper treatment, to safeguard the vision, and to shorten hospitalization. As a result of uncontrolled or long persisting papilledema permanent damage to the vision may develop rather suddenly in these cases.

Photophobia is a frequent symptom, as is headache which may be frontal, occipital, or diffuse. Vomiting is usually of the projectile type, and Cushing[11] says that vomiting is one of the most persistent and earliest signs of hydrocephalus. Nystagmus and vertigo have been attributed to endolymphatic pressure as they promptly disappear on lowering the cerebrospinal pressure. The pulse rate may be slow or fast depending upon irritative toxins or the slowing effect of intracranial pressure. When pressure is high, torpor, indifference, or stupor may ensue. Reflexes may be exaggerated, diminished, or absent. Meningitic signs are not frequent except in acute aseptic meningitis. While the findings are few, brain abscess is very frequently suspected in the patient with otorhinogenic hydrocephalus.

The differential diagnosis is often difficult, especially in the early stages. For this reason and in suspected septic meningitis, Williams states that complete removal of an exciting focus should be done in any patient exhibiting signs of meningeal irritation. Acute lymphocytic meningitis should present a large increase in mononuclear cells in the spinal fluid in the early stages. However, if associated with otitis, differential diagnosis might be impossible. Tuberculous meningitis should present maximum meningeal signs, low cell count, low sugar and high spinal fluid chlorides which are said to be almost diagnostic. Lateral sinus thrombophlebitis is in itself one of the most frequent causes of otitic hydrocephalus. And finally brain abscess is probably the most often suggested by the symptomatology, and is commonly the most difficult to differentiate. It is significant that in the 60 cases of phlebitis of the lateral sinus reported from the St. Louis Children's Hospital between 1920 and 1936, Hartmann and Cone[12] found that papilledema with or without demonstrable increase in spinal fluid pressure occurred in 41 per cent of the cases in which papilledema was looked for. Brain abscess was frequently suspected but apparently did not occur in any of their cases.

Williams and others, however, have reported cases of otitic hydrocephalus and brain abscess. Williams points out the extreme difficulty in differentiating otitic hydrocephalus from brain abscess if a preliminary stage of meningitis has caused a temporary excess of cells and protein in spinal fluid with persisting pleocytosis. On the other hand, lack of pleocytosis may be the only finding suggesting that brain abscess is not present. Differential diagnosis may require diagnostic surgery or ventriculography. When necessary, foci should be eliminated in an orderly manner, and this may include mastoidectomy and extensive exposure of the dura or comparable sinus surgery. Exploration for brain abscess is considered justifiable under certain

conditions, if extradural or subdural abscess has not been found. It is unfortunate that brain abscess must be ruled out before proper treatment can be safely instituted to control the otorhinogenic hydrocephalus.

Treatment, in addition to fundamental, well-established pediatric and medical principles, includes removal of any exciting focus as mentioned previously, and reduction of cerebrospinal fluid pressure by systemic means and by removal of excessive fluid until such a time has elapsed that an adjustment has been effected to again maintain the normal pressure. Hypertonic fluids exert only a transient influence. Frequent small transfusions as suggested by Kopetsky[13] help to keep down the pressure by the effect on the electrolytes and the lactic acid of the spinal fluid. Dehydration and limitation of fluid intake was an important factor in the case reported here. Therapeutic lumbar punctures have been invaluable for those with communicating hydrocephalus.

Symonds felt that lumbar puncture was justifiable in the hypertensive state because obstructive internal hydrocephalus is so rare. Extreme caution has been urged if brain abscess is suggested. Many have pointed out the risk of lumbar puncture because of the danger that a brain abscess is present. It is significant that the one case of otitic hydrocephalus from the St. Louis Children's Hospital who did not receive therapeutic lumbar punctures for that very danger, the possibility that a brain abscess was present, developed optic atrophy and total blindness. Dean, who strongly advocated therapeutic lumbar punctures for this condition, maintained that the danger of this therapy was slight as compared to the danger of blindness from uncontrolled papilledema, and cited two cases of total blindness where lumbar punctures were denied for the hypertensive state. If lumbar puncture is contraindicated, ventricular puncture and decompression may be done in greater safety. The important indication is to reduce the intracranial hypertension until a proper adjustment can develop to maintain the normal spinal fluid pressure, although a number of weeks may elapse before inflammatory subependymal changes decrease and collateral venous circulation develop.

By reviewing the complications encountered in caring for D. H., a 7-year-old girl, who survived a severe otitic hydrocephalus following mastoiditis, brain abscess, meningitis, and probable lateral sinus thrombophlebitis, it is hoped to emphasize the clinical picture, differential diagnosis, and therapeutic problems of this condition.

This case was reported in 1942 as one of a group of severe infections in children by Hartmann, Wolff, and Love.[14]

REPORT OF A CASE

Following pneumonia at six months of age, otitis media first developed. With colds and each succeeding winter there would be a recurrence of ear pain, fever, and purulent discharge from the left ear. Her parents had noticed a progressive loss of hearing. About one month before admission she again complained of severe left earache and developed a persistent fever which reached 103.5° F. There were no chills. Two weeks before admission a severe frontal headache began. About the same time spontaneous ear discharge occurred, but the fever persisted. For three days before being referred by her local physician to St. Louis Children's Hospital she had taken practically nothing by mouth, and had responded only to the most insistent demands.

On admission on February 22, 1940, all of the above conditions were present, and in addition there were severe dehydration, emaciation, and anemia. Temperature was 101.5° F., pulse was 120, and respirations were 20 per minute. There was a fine nystagmus to the right. The pupils were dilated but reacted to light. The fundi showed only suggestive haziness of the left disk. Foul pus pulsated through a mass of red granulations in the depth of the left canal. The right ear was dry and the drum was dull and thickened. There was a slight left central facial weakness. The lungs were clear to physical and fluoroscopic examinations. The heart was within normal size but had a loud systolic murmur. Knee kicks were brisk; ankle clonus was present on the left. The original impression of Dr. Alfred Schwartz, who admitted her, was that she had a left mastoiditis, localized meningitis, probably a left cerebellar abscess, and possibly a lateral sinus thrombophlebitis, all of which were later found to be present. A diagnostic lumbar puncture revealed ground-glass appearing fluid with approximately 1000 polymorphonuclear cells, 3 plus Pandy, and gram-positive cocci which failed to grow in the laboratory. The Tobey-Ayer test was positive on the left. Dehydration was quickly relieved by parenteral fluid. Chemotherapy was started with intravenous sodium methylthiazole. Urine and Kline tests were negative. White blood count was 19,000 with 86 per cent polymorphonuclears. Shick and tuberculin tests were positive. X-ray examination revealed extensive destruction of the left mastoid with a well defined rarefied area in the region of the knee of the lateral sinus, which was interpreted as a probable perisinus abscess.

By the afternoon of the following day the Pediatric Department considered her condition satisfactory for an exploratory left mastoid operation. The sterile character of the meningitis had been determined, hydration and sulfonamide concentration were good, and a transfusion had been given. It had been decided in consultation with Professors Alexis F. Hartmann and L. W. Dean that, in order to save time, it would be advisable to ligate the left internal jugular vein before opening the mastoid. The vein was thick and grey in the midportion of the neck, but looked normal at a lower level. Subperiosteal abscess, large confluent mastoid abscess, extensive bone destruction, and cholesteatoma in the antrum were encountered. The lateral sinus region was covered with extensive granulations. The middle fossa dura was thick and reddened. In spite of extensive exposure of the middle and posterior fossa dura, normal appearing structures could not be found, and it was not possible to identify the lateral sinus. A posterior fossa abscess, containing approximately two ounces of foul pus, was suddenly encountered while removing necrotic bone deep in Troutman's triangle. A rubber dam drain was placed into the abscess, and the mastoid wound was packed open. Bacillus Proteus was grown from the mastoid secretion, and Bacillus Aerogenes was cultured from the brain abscess.

Following the operation and with continued intravenous sulfamethylthiazole and supportive treatment, gradual improvement occurred. Two days later she talked a little. By February 28 she was taking a soft diet, and chemotherapy was continued by mouth. On March 3 the Department of Ophthalmology reported normal fundi. March 12 the drain was completely removed from the posterior fossa abscess after gradual shortening at each of the daily dressings. On March 14 she complained of frontal headache, and papilledema of about one diopter was present bilaterally. Daily therapeutic lumbar punctures done carefully with a manometer reduced the papilledema, and the headache disappeared. Chemotherapy was discontinued March 22 when temperature, pulse and respirations had been normal for three weeks. She was moved from her room into the ward to be with the other children at her request.

One week later she became irritable again, complaining of headache and photophobia. Papilledema was again evident, and she vomited four times. Spinal fluid pressure had risen to 200 mm. of water, and there were 65 cells. Because of this the mastoid was re-explored on March 30 with the hope of replacing the drain in the posterior fossa abscess which was thought to be reforming, and to again at-

tempt to identify and drain the lateral sinus. With the assistance and counsel of Dr. A. J. Cone this procedure was carried out 35 days after the original operation. The dura was exposed again very widely and was found to be solidly healed in the region of the previous spontaneous drainage from the posterior fossa. Again the lateral sinus could not be identified. However, there was a soft, yellow area in the sinus region. This was incised and necrotic tissue was obtained with a microscopic picture of chronic encephalitis. Spinal fluid seeped through the 7 mm. opening in the dura until this opening was blocked several days later by a cerebellar herniation.

Two days later spinal fluid cell count had increased to approximately 2000. Chemotherapy was started again, this time with sulfapyridine as sulfamethylthiazole had been withdrawn from use. Spinal fluid cultures were negative as before, and the cell count dropped promptly. In spite of this, papilledema was increasing, and spinal fluid pressure reached as high as 295 mm. of water. She became more stuporous as the cerebellar herniation reached the size of a walnut. On April 14 there was definite indication that her vision was poor, the herniation was larger, and she again turned away from light. On April 16 pneumo-encephalograms showed marked symmetrical dilatation of the ventricular system without evidence of brain abscess. Although definite temporary improvement followed each of the daily lumbar taps, the cerebellar herniation slowly increased, and the spinal fluid pressure varied from 220 to 300 mm. Accordingly the therapeutic lumbar punctures were increased to twice daily. From 80 to 95 cc. were removed each time using a manometer to reduce the pressure to normal.

On April 29 her fluid intake was restricted to 500 cc. daily. After May 8 the herniation did not enlarge. She could tell night from day although the disks were pale and still moderately elevated. The cerebellar herniation was reduced with diathermy after which the dural opening healed. On May 28 she was up in a wheel chair. The postauricular wound was healed. In June the lumbar taps were reduced to once daily and then to every second day. There was moderate left-sided incoordination. Vision was improving. Papilledema disappeared by the middle of June. Although spinal taps could be discontinued it was necessary to continue the limitation of fluid intake to avoid bulging of the postauricular bony defect.

She was discharged from the hospital on July 5 after a stay of 104 days. She returned to school that fall. In October 1940 she seemed perfectly well. Her vision was good, the disks were pink and

without elevation, and her gait was fairly normal. The following year her mother reported her continued good health and progress in school.

DISCUSSION

The reported case illustrates the clinical picture of otorhinogenic hydrocephalus. The difficulty in differential diagnosis, especially from brain abscess, is of the utmost importance since the proper treatment depends vitally upon the correct diagnosis. An increasing incidence of this hypertensive intracranial state should be expected with increasing recoveries that will follow the greater use of sulfonamides, penicillin and future chemotherapeutic agents. It is suggested that this complication should be anticipated in any severe infection of otitic or sinus origin. Rather extreme measures were required to control the abnormally high spinal fluid pressure until compensatory changes could develop in this patient. Recovery with serviceable vision more than justified the therapeutic risks when the danger of total blindness without adequate treatment is considered.

STATION HOSPITAL.

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LXVII

TREATMENT OF INOPERABLE CANCER OF THE THROAT

FOLLOW-UP OF PREVIOUS REPORT[1]

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It is the purpose of this paper to report our experience in the treatment of far advanced or late cancer of the throat by an improved method of approach. There are many points of interest and importance in connection with our experience with this group of cases, but at least two facts stand out pre-eminently. First, although the disease in these patients had progressed to the very late stages, viz., widespread involvement of the larynx and pharynx, the base of the tongue and the glands of the neck with, of course, marked interference with the functions of respiration, phonation and deglutition, and with anemia, cachexia and weight loss, many of them are alive and free of evidence of cancer, some for as long as four years. Second, they were treated according to a plan new to us, which we worked out in attempting to avoid x-ray injury to the thyroid cartilage and the deadly complications and sequelae which follow such injury.

The disastrous effects of radiation injury to the laryngeal cartilages have been recognized by all workers and fully described in the literature and because of this difficulty with the laryngeal cartilages, radiation therapy has been discarded by many as a means of treating any but hopeless cases.

Thomson[2] in an article published in 1926 discussed radiation therapy as a palliative measure to be "attempted only in hopeless cases," calling attention to delayed reaction one to six years afterwards resulting in "ulceration, necrosing, and death within a short time." The same author in his book *Cancer of the Larynx*,[3] published in 1930, in the chapter "Treatment by Deep X-rays", which

is resplendent in bibliography, calls attention to "special dangers in the application of x-rays to the larynx, which has proved itself to be peculiarly sensitive to their action".

Other articles of particular interest in this connection are those by Iglauer,[4] Levitt,[5] Clerf,[6] Strandberg[7] and Watt,[8]

Hautant[9] observed that when necrosis follows irradiation the sequestrum is triangular in shape and that it consists of the ossified portion of the thyroid cartilage at the anterior-inferior angle of each wing; therefore, Hautant now resects this portion of the thyroid cartilage by a purely external operation as a preliminary to x-ray treatment and holds the operation of cutting a window in the middle of the wing in disregard. Flurin and Magdeleine[10] suggest, in addition to these precautions, that the growth should be excised, thus converting the use of x-rays into a prophylactic against recurrence rather than employing it as the chief weapon for destroying the growth. Those workers were able to present encouraging results in x-ray treatment of endolaryngeal cancer to the International Congress of Otology in 1922, and Hautant was able to report in 1927 three of the six patients had remained well.

Thomson remarks on the many factors affecting the results, such, for example, as the histological character, the degree of vascularity, the character of the growth, whether it is nodular or ulcerating, its extent, whether it is limited to a cord or infiltrating muscle, all of which are of importance. After further observations on the varying response according to cellular type of the growth and the quality versus the quantity of the rays, Thomson says, "It is clear that if the preliminary resection of cartilage is performed by the method of Hautant, a proportion of permanent results may be obtained". He further says, "Radiation should be reserved for cases with indications which render them unsuitable for surgery." He quotes MacLenty, Tucker and others, who prefer surgical intervention to irradiation.

It is plainly evident that in the type of cases discussed in this paper, hope of successful surgical removal is out of the question; therefore, the only treatment offering any hope is irradiation, and this hope of cure is marred by the expectation of almost certain injury to the thyroid cartilages.

We have been able to show in this small group of cases of far advanced cancer of the throat that by preliminary removal of the entire thyroid cartilage, we may carry out x-ray therapy in dosage

adequate to kill the tumor cells, and, furthermore, after the reaction has cleared up, the patient may expect to have almost normal respiration, phonation and deglutition. He is able to follow a useful vocation and live normally.

The patients whose cases are described in this paper were found on the wards of the county and city hospitals and in our private practice. As has been said above, all were typical late generalized cancer of the throat with glandular involvement, cachexia, and interference with deglutition, respiration and phonation, and with the usual pain and weight loss, without possible hope of cure by surgical means. In some, the original site of the lesion had been in the larynx; in others in the epiglottis; in still others at the base of the tongue or in the laryngopharynx. In one or two the cancer apparently had started in the hypopharynx, and in one woman the lesion involved and blocked the mouth of the esophagus. In nearly all cases the cancer had spread to the neck, including the lymphatic and the thyroid glands. Most of the males had their share of constitutional diseases seen in individuals of this age group, such, for example, as cardiovascular-renal disease or diabetes. These proved to be added hindrances to treatment and, because of their severity, at times rendered maintenance of life difficult. The mortality rate has been surprisingly low, we think, and furthermore, the suffering and the general effects of treatment have been comparatively less severe than was previously our experience.

Since the life expectancy of individuals with cancer of the throat in the late stages may be measured in weeks, or at best, a very few months of misery and utter hopelessness as to prospect of relief or cure, the contrast in this group needs no comment. We are, of course, conversant with the surgical treatment of cordal cancer, and we are in full accord with the teachings of Thomson regarding the importance of proper selection of cases of cordal cancer for surgery, as well as with the results obtainable by this method of treatment. We have also had experience with laryngectomy for further advanced cancer, but the cases under discussion in this paper do not fall in the group in which surgical removal is possible.

It seems to be generally conceded that the only possible hope of relief in this type of cancer of the throat is by irradiation therapy, and this has generally been considered as palliative rather than curative. With dosage sufficient to kill the cancer, x-ray injury to the cartilage was highly probable. Then necrosis and infection of the laryngeal cartilages spread by contiguity into the neighboring tissues, finally opening the great vessels of the neck. Inadequate dosage was

followed by recurrence or what was more probable continuation of the cancer.

Having learned that the laryngeal functions are not materially impaired by total removal of the thyroid cartilages, one of us (MFA) decided, in order to be free of the complications which follow cartilage injury by radiation, to excise the cartilages in toto before beginning radiation. After the diagnosis had been proven microscopically, no attempt whatever was made to excise the tumor or any part of it. We discovered later that the cartilage is replaced within a few months by a firm, fibrous box, apparently from the perichondrium. We have as yet not been able to examine a specimen from one of these larynges after it had healed.

We knew the thyroid cartilage often was killed by x-ray, but we were not sure about the other cartilages. Unfortunately, the available literature gives very little information on the effects of radiation on cartilage. In a series of very interesting and informative articles a few years ago, Desjardins[11] described in detail the effect of radiation on the tissues of the human body with the exception of the cartilage. One of us (MFA) planted gold seeds in the costal cartilages of dogs and studied the tissues in the immediate vicinity of the seeds afterwards with negative results.

We had seen evidence of reaction in the epiglottic cartilage, a sort of brawny edema, so, at first, we removed the thyroid and epiglottic cartilages, subperichondrially; later on we removed only the thyroid cartilage. While there was reaction in the epiglottis, the same brawny edema, in some (not all) patients, this soon cleared up. Anesthesia consisted of avertin, given locally and then intratracheally by means of a catheter through the tracheal opening. Incision for removal of the thyroid cartilage was made in the midline from the level of the hyoid bone down to the fourth tracheal ring. A tracheotomy tube was inserted through the third and fourth rings. The outer perichondrium and the thyroid cartilages were divided in the midline, with care not to open the inner perichondrium, and complete subperichondrial elevation, outer and inner, was made. The cartilage was then lifted out with forceps and the wound closed in layers. A feeding tube through the nose was left in position. Great care should be exercised not to open the inner perichondrium and mucous membrane in order to avoid infection in the wound.

Injury to the cricoid cartilage should be avoided. This requires care while splitting the partially calcified thyroid cartilage. In one case while we were using much pressure to incise the thyroid cartilage,

which was dense and brittle, the cartilage cracked and the knife plunged into the cricoid cartilage below. The soft tissues healed within the usual eight or ten days, but evidence of infection of the cricoid appeared after irradiation and persisted until some weeks later. Then through a midline incision we found a fistula at the point of the knife injury, leading into the marrow space, as it were, and involving the entire cricoid cartilage. The cartilage was destroyed and has contracted but the man has a fair voice and works every day, as of July 26, 1944. The x-ray treatment was given according to the Coutard technique, and the fullest possible dosage was maintained in each case until the prescribed course had been completed.

Close cooperation between the surgeon and the radiologist is imperative in order that the dosage of x-ray may be properly controlled. This is controlled by local signs, usually referred to as mucositis and epidermitis; and the constitutional reaction, as is evidenced by vomiting, prostration, dehydration, failure of appetite, rapid weight loss, or exacerbation of serious constitutional disease. Ability to recognize the changes in the mucous membrane resulting from irradiation is gained only by experience. This control means daily or almost daily mirror examinations of the throat, as well as examination of the neck, consideration of the general reaction with regard to nausea, attention to the intake and output and the need for relief of pain. The status of constitutional disease of one or another variety, which is so often present, is evaluated and controlled by an internist, and an internist should always be one of the group working together in the treatment of these patients.

We feel that dosage sufficient to kill cancer cells is invariably accompanied by skin burns and x-ray sickness. This is of greater severity in some individuals than in others, and because of x-ray sickness, it is sometimes necessary to diminish or interrupt the x-ray treatment for a short period. Occasionally unfavorable reactions in patients suffering with cardiac disease or diabetes render it necessary to discontinue treatment temporarily. In such cases, with proper medical care, the results have proven satisfactory.

Since the suffering which accompanies treatment usually is severe, it is our practice to warn patients in advance of the trials to which they may be subjected during the course of x-ray treatment.

The accompanying tables show the number of cases with essential data in each. All cases are proven microscopically and graded according to the degree of cellular differentiation. We found all types, including well differentiated and poorly differentiated (1 to

4) present. The type apparently had no influence on the response to irradiation. No attempt at surgical removal of the tumor was made.

SUMMARY

Our experience in the treatment of 16 cases of inoperable cancer of the throat by an improved mode of approach is herewith reported. Eight are living and free of signs of cancer. All have normal deglutition and a good voice. Nearly all have a normal airway, but wear a small-sized tracheotomy tube which they keep plugged.

Emphasis is placed on the importance of a new fundamental principle in the treatment of advanced cancer of the throat. The principle is founded on the removal of the thyroid cartilage before x-ray therapy is instituted. In our preliminary report of this work, we did not give credit to Hautant[9] for his work for the reason that we were unaware of it until July 12, 1944, for which we apologize.

Attention is further called to the need for adequate dosage, careful evaluation of the x-ray reaction, and supervision of accompanying generalized disease. The possibility of extending this form of treatment to earlier cancer may be considered.

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TABLE 1.

Case No.	Age	Sex	Clinical & Microscopic Diagnosiss	Date of removal of Thyroid Cartilage
1. A. S.	57	Male	Squamous Cell Carcinoma	5-8-40
2. A. W.	56	Male	Squamous Cell Carcinoma	10-8-40
3. W. K.	71	Male	Squamous Cell Carcinoma	8-6-40
4. W. Q.	63	Male	Squamous Cell Carcinoma	11-18-40
5. J. D.	54	Male	Squamous Cell Carcinoma	1-5-42
6. J. W.	62	Male	Squamous Cell Carcinoma	8-5-43
7. J. B.	61	Male	Squamous Cell Carcinoma	9-25-43
8. J. P.	70	Male	Squamous Cell Carcinoma	8-26-43
9. G. M.	55	Male	Squamous Cell Carcinoma	11-30-43
10. E. B.	63	Female	Squamous Cell Carcinoma	9-28-41
11. A. M.	60	Male	Squamous Cell Carcinoma	4-29-44
12. E. S.	53	Female	Squamous Cell Carcinoma	12-4-33
13. H. J.	....	Female	Squamous Cell Carcinoma	12-28-40
14. E. B.	28	Female	Squamous Cell Carcinoma	1-6-36
15. C. H.	75	Male	Squamous Cell Carcinoma	Records not available
16. B. H.	68	Male	Squamous Cell Carcinoma	Records not available
17. J. H.	70	Male	Squamous Cell Carcinoma	Records not available
18. T. O.	56	Male	Squamous Cell Carcinoma	Records not available

TABLE 2.

Case No.:	Date X-ray Treatment Started:	Date X-ray Treatment Concluded:	Date and Results of Last Follow-up Examination:
1. A. S.	5-29-40	6-9-40	5-8-44: No evidence of cancer, is active and able to work.
2. A. W.	10-21-40	12-2-40	7-26-44: No evidence of cancer, working every day.
3. W. K.	8-26-40	.....	Died.
4. W. Q.	11-28-40	.....	Died March 1942.
5. J. D.	1-19-42	2-5-42	7-24-44: Working every day, no sign of cancer.
6. J. W.	8-17-43	9-17-43	7-12-44: Working every day, no sign of cancer.
7. J. B.	10-25-43	11-20-43	Died May 1944.
8. J. P.	9-9-43	9-29-43	Living and well.
9. G. M.	12-9-43	12-23-43	Died June 1944 of strangulated hernia but no cancer found at postmortem.
10. E. B.	10-8-41	.....	Died Nov. 1941.
11. A. M.	10-10-43	11-1-43	Living and well. Radon to gland in neck.
12. E. S.	12-14-33	1-1-34	Died 1943.
13. H. J.	1-8-41	1-28-41	Living and well but with stricture of esophagus.
14. E. B.	1-16-36	2-5-36	Living and well.
15. C. H.	Records not available		Died.
16. B. H.	Records not available		Died.
17. J. H.	Records not available		Died.
18. T. O.	Records not available		Died.

TABLE 3.

AMOUNT OF RADIATION GIVEN IN SOME CASES:

Case No. 1.	A. S.	2400 R. U. to each side of neck (300 a day) May 20 to June 9, 1940, inclusive. Barnes Hospital.
Case No. 2.	A. W.	3600 R. U. to each side of neck (200 a day) Oct. 21 to Dec. 2, 1940, inclusive. Barnes Hospital.
Case No. 5.	J. D.	2100 R. U. to each side of neck, Grade 2, Jan. 19 to Feb. 5, 1942. St. Lukes Hospital.
Case No. 6.	J. W.	4800 R. U. over a period of four weeks. Operated on Aug. 5, 1943. Jewish Hospital.
Case No. 7.	J. B.	2800 R. U. to each side of neck, Oct. 25 to Nov. 20, 1943. Barnes Hospital.
Case No. 8.	J. P.	2400 R. U. to each side of neck, Sept. 9 to Sept. 29, 1943. Barnes Hospital.
Case No. 9.	G. M.	200 R. U. to each side of neck on alternate days, from Dec. 9 to Dec. 22, 1943, inclusive, a total of 1200 R. U. given. City Hospital.

LXVIII

AN UNUSUAL NEOPLASM IN THE LARYNX OF A CHILD (RHABDOMYOMYXOSARCOMA)

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In the practice of the average laryngologist sarcoma of the larynx is rarely or never encountered. This is particularly true in the case of an infant or child, since no actual report of its occurrence in the very young could be found from a review of some of the older and also the more recent textbooks of pathology and the literature prior to 1943. A report of the following case is, therefore, submitted because of its rarity and also for its clinical interest from the standpoint of laryngeal findings, management and subsequent progress.

REPORT OF A CASE

R. S., a 10-year-old white, school boy, came under my care on January 18, 1940, because of difficulty in breathing for two weeks and increasing hoarseness of one year's duration. The boy's father, who accompanied him, stated that he thought the trouble in the throat started while his son was playing soldier and making noises like the firing of a machine gun. He gave no history of injury, and as far as he knew, the boy's health was fairly good with the exception of an occasional sore throat. In May 1939, he took him to a hospital clinic where he had his tonsils and adenoids removed. For some time after the operation, attacks of sore throat were less frequent but there was no improvement in his hoarseness. Because his breathing was gradually getting worse, especially at night, and also because he was starting to have some trouble with swallowing at meal times, the father consulted his family physician who advised immediate laryngological care. On the basis of this history, an examination of the larynx was promptly made and the patient hospitalized.

Mirror examination of the larynx was easily carried out. The base of the tongue, the epiglottis, larynx and surrounding parts were exposed. There was slight hypertrophy of the lingual tonsils but no redness. The epiglottis was normal in shape and slightly overhanging

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but did not interfere with a good view of the larynx. The aryepiglottic folds and the arytenoids, as well as the pyriform sinuses, appeared normal. The major portion of the laryngeal opening was filled with a smooth, soft, grayish-looking tumor mass, extending from the anterior commissure slightly below the left cord surface. This was approximately the size of a small lima bean.

After the examination disclosed the presence of a tumor mass in the larynx interfering with the boy's breathing, he was admitted to the St. Louis Children's Hospital. He appeared fairly comfortable when he entered, but it was evident that his breathing was noisy but not labored. His general physical condition did not reveal any findings of importance. The family history was essentially negative—there was no tuberculosis, asthma, hay fever or cancer. He had measles at 18 months, scarlet fever at two years, chicken pox at four years, whooping cough at five years, mumps at eight years, and tonsils and adenoids removed at nine years. There were no sequelae. There was no history of cough, hemoptysis, headache, nausea, vomiting, diarrhea, abdominal pain, colds or discharging ears.

Shortly after admission to the hospital, his dyspnea increased and some chest retraction appeared during respiration. A tracheotomy was promptly performed under local one per cent novocaine anesthesia. The thyroid isthmus was cut and ligated. The second and third tracheal rings were incised and a No. 3 cannula inserted. Following the insertion of the tracheal cannula, the laryngeal obstruction was completely relieved and a direct examination of the larynx was made with the Jackson laryngoscope.

The laryngoscopic examination showed the laryngopharynx slightly injected; the epiglottis normal in shape and structure, with small, overhanging, curved upper edge. The aryepiglottic folds, arytenoids and ventricular bands showed some thickening probably due to mild edema. The true vocal cords appeared chronically injected. A firm, smooth, grayish-blue subglottic mass was exposed which nearly filled the glottis. A free rounded edge could be made out in front of the posterior commissure, and the mass seemed to spring from the left cord margin as far as the anterior commissure. No specimen for biopsy was taken at this time and no attempt was made to remove the tumor. A suspension laryngoscopy was considered as a necessary subsequent procedure in order to give a better opportunity for the study of the character of the lesion and its surgical management.

On January 22, 1940, four days after admission to the hospital, a Lynch suspension laryngoscopy was performed under ether anes-

thesia. The larynx was exposed easily and the tumor mass could be seen to be smooth and firm in appearance. On palpation with a laryngeal probe, it did not prove to be freely movable. It filled the laryngeal opening and was attached to the left side of the glottis along the true cord. On further examination, it proved to be more subglottic in character, extending from the upper surface and inner edge of the left cord and reaching along the entire anterior two-thirds of its length to the anterior commissure. It was grayish-blue in appearance and it was difficult to determine if it was imbedded in the substance of the cord. To establish the diagnosis of this tumor and the character of its treatment, a specimen for biopsy was taken from the mass with punch forceps and sent to the pathological laboratory. Very little bleeding followed. An attempt was then made with grasping forceps to deliver the remaining portion of the tumor in front of its free end and to determine if it could be brought out above the surface of the cords. If this were possible, it was reasoned that the tumor could be removed completely with a snare, after freeing its attachment to the cord by dissection. This did not prove successful because the tumor appeared to be imbedded in the cord structure. The major portion of the tumor up to the margin of the cord was then removed with punch forceps. The tissue at first cut as if it were soft, but in the area below the cord level the tissue was firm and cut harder. A very good laryngeal opening was obtained between the cords. Since it was not possible to remove all the tumor tissue in the cord and below it satisfactorily at this time, an external operation by the laryngofissure route was decided upon at a later date.

Microscopic examination of the tumor tissue removed from the larynx during suspension laryngoscopy was described by Dr. L. W. Dean, Jr., of the Department of Otolaryngology, Washington University Medical School, and reported as follows: "Tissue is made up of cells typical of myxoma, i. e., thin stellate cells with long narrow processes which interlace. Diagnosis—Myxoma." Inasmuch as the tissue removed from the larynx was an unusual example of a neoplasm in a child, it was submitted for further study, particularly to determine the possibility of malignancy.

In order to remove the tumor from the larynx completely, an external operation was performed on January 29, 1940, under intratracheal ether anesthesia. The larynx was opened in the midline of the neck, the incision extending from below the hyoid to the tracheal fistula. The thyroid cartilage was incised with a scalpel and included a small upward incision into the thyrohyoid membrane and through the cricothyroid membrane. The right cord was found normal and

the tumor tissue about the left true cord seemed to occupy a larger area below the cord than seen during the examination by suspension. It definitely appeared to be an integral part of the cord. The inner perichondrium of the left thyroid ala was elevated above and below the true cord area, extending from above the false cord to the upper margin of the cricoid cartilage and posteriorly to the vocal process on that side. It was removed in one mass with a nasal snare and sent to the pathological laboratory for study. There was no bleeding when the mass was cut from the inner laryngeal wall except when it was completely detached near the arytenoid. This bleeding was apparently from a small internal branch of the superior laryngeal artery on that side which was controlled with one suture ligature.

The wound was closed, allowing the thyroid cartilages to fall in their normal position in the midline; mattress sutures were used, one above and one below the cartilage margins to hold the membranes together. Subcutaneous sutures were used to eliminate pockets and the skin closed with silkworm gut and clips. The patient was returned to his room in good condition and subsequently made a good recovery. On February 2, the clips and sutures were removed, and on the following day the tracheal cannula was removed, allowing the fistula in the neck to close.

The boy seemed to be doing very well for about four weeks after the laryngofissure, when one day he suddenly developed a severe dyspnea and had to have another tracheotomy performed. On the following day, under suspension laryngoscopy, I removed a collection of flabby loose tissue from the previously operated area, which proved to be granulation tissue. After this experience his cannula was not removed. He wore it with the tube opening covered, so that an airway could be assured if subsequent laryngeal obstruction developed. After several weeks the tube was removed and he has done very well since that time. His health has improved, he shows no evidence of recurrence, his voice is fairly strong, and he has no respiratory disturbance. He now can play as hard as any other boy.

COMMENT

The occurrence of the tumor in the boy's larynx which, upon removal and microscopic study, proved to be a mixed form of sarcoma is most unusual. Nearly all writers in the reports from the laryngological literature during the past 50 years agree that true sarcoma is uncommon and, when encountered in the larynx, occurs in adults, is chiefly intrinsic, shows little tendency to metastasize early, especially at the site of removal, and is found relatively more frequent-

ly in males. Lederer[1] states that according to some statistics it occurs in less than one per cent of all malignant laryngeal neoplasms, grows more slowly than carcinoma, and has a predilection for the vocal cords.

In a review of the Transactions of the American Laryngological, Rhinological and Otological Society, as far back as 1897, only a few cases are mentioned. In 1933 Figi[2] reported four cases of sarcoma of the larynx out of 713 laryngeal neoplasms seen at the Mayo Clinic since 1910. He also mentions that in Jackson's series of 643 laryngeal neoplasms, not one case of true sarcoma was encountered, and in the wide experience of Thomson and Colledge only one case was seen. It is interesting to note also that in 700 cases at the Mayo Clinic, extending over a period of 30 years, a very small percentage of pure myxomata was encountered. New[3] in 1935 and Havens and Parkhill[4] in 1941 reported additional cases of laryngeal sarcomas. The only record that I have found of a malignant tumor occurring in an infant, which proved on postmortem study to be a fibrosarcoma of the larynx, was reported recently by Rigby and Holinger.[5]

Since the histologic structure of these sarcomatous tumors sometimes show wide variations because of tissue origin, it may be possible for errors in diagnosis to be made. This may explain the fact that fewer true sarcomas are reported. Most pathologists emphasize that a diagnosis is only possible with a careful microscopic study of the tumor itself. It is apparent, however, from the study of this case and the discussions that developed, that there is discord among pathologists in arriving at a diagnosis. Perhaps this has led to the opinion held by some laryngologists that tumors of this nature are histologically malignant but clinically benign. Time alone is the important factor; the treatment of these cases according to most authorities depends on the size, situation and activity of the tumor growth.

For this patient who is getting along so well no decision has been reached as to any further management. It had been suggested sometime ago that a laryngectomy should have been performed, but I was very reluctant to do so. In fact, one shudders at the thought of such an operation in a child when the necessity is in doubt. As a matter of fact, I have never found a record nor heard of a case of a laryngectomy in a child. With the diagnosis of actual malignancy still subject to question and with the boy's health improving, nothing further is contemplated at the present time.

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LXIX

AUDITORY ACUITY OF AVIATION CADETS

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There has recently appeared a series of reports from the Services describing the incidence and character of hearing defects found among officers and enlisted men returning from combat. Of necessity, these observations have consisted of audiometric studies performed after exposure to aircraft noise, gunfire, or barotrauma without precombat auditory thresholds.

No controlled studies of so-called war deafness are available in the literature at the present time. In order to evaluate hearing losses among army personnel, it is essential to know the types and grades of hearing deficiencies occurring among men who have had no combat service.

During World War I, Guild[1] noted the increasing incidence of auditory damage. Campbell,[2] Dickson,[3] and Bunch,[4] among others, called attention to the deleterious effect upon the organ of Corti of repeated and prolonged exposure to aircraft noise of high intensity.

Several good audiometric surveys have been made of the civilian population. Bunch[5] classified 353 hospital patients according to age and demonstrated the progressive elevation of high tone thresholds with increasing age. Montgomery[6] found corroborating data among 200 "normal" individuals. Rodin,[7] using the Western Electric 4A audiometer, studied 36,191 school children (9-16 years of age) and found that 9.5 per cent had losses of nine or more sensation units in one or both ears.

In a National Health Survey, Beasley[8] and his associates observed 4,364 persons (ages 8 to 76 years) who were grouped on the basis of a clinical interview as falling into a broad classification of individuals free of any of the arbitrary "stages of deafness." Audiograms were obtained with the Western Electric 2A audiometer and those showing losses of 20 db. or less over the eight tones tested were considered

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"normal." Only 38 per cent of this group had "normal" hearing in both ears.

Crowe, Guild, et al,[9] in a study of 672 boys (8-14 years of age), found that less than 47.3 per cent of the group had "normal hearing" when tested with the Western Electric 1A audiometer (32-16,384 cps); 29.0 per cent had bilateral impairment for high tones; 18.0 percent had one normal and one deficient ear; 3.0 per cent showed a hearing impairment of 9 per cent or more using one or both ears.

In a review of these same 672 boys, Loch[10] found that 15 per cent of the ears showed tonal dips. These tended to occur three times more often at 4096 cps than at any other frequency and were more prevalent in older children.

Dickson, Ewing and Littler[11] stated that even after a few hundred hours of flight there was a persistent loss of hearing for high tones which was evidenced at first by a dip at 4096 cps. They note that this "aviator's deafness must be considered as potentially threatening a serious and lifelong disability."

Campbell and Hargreaves[12] demonstrated that repeated exposure to aircraft noise may cause a tonal dip in the 4096 area and, depending upon individual susceptibility, may result in a fanwise spread of the notch so that there is difficulty in hearing conversational speech.

Elder,[13] studying auditory thresholds on three young chimpanzees, found a marked and unexplained dip at 4096 cps which was most pronounced in the oldest animal.

Procedure. Five hundred healthy males between the ages of 18 and 27 years (mean age, 21.1 years) were selected at random from among the enlisted trainees at a classification center (San Antonio Aviation Cadet Center). These men had passed Class I requirements of the W.D. AGO 64 examination. All subjects had had little or no flying experience and had not been exposed to aircraft noise for approximately 30 days.

Auditory threshold tests were performed in sound-conditioned or sound isolated rooms in which there was 38 db. or less of ambient noise (General Radio 759-B Sound Level Meter, "A" weighting). The three Maico D-5 audiometers used in the survey had been checked for relative sound pressure and voltage output, accuracy of attenuation and purity of sine wave. All audiometers were calibrated against a General Radio Sound Level Meter, using a small volume

coupler technique, and did not deviate more than plus or minus 5 db. from each other at any of the nine frequencies used.

Five examiners (SN, OG, AN, SU, and MC) performed the auditory threshold tests. Each was given a preliminary period of instruction, sufficient training to insure reliable results and was supervised to assure a uniform technique of test presentation. Periodic checks were performed from time to time by the writer to insure accuracy of the threshold curves.

Audiograms were typed and graded by one observer in accordance with a system previously outlined.[14] The results were classified into four types on the basis of the configuration of the threshold curve, viz., A, V, W, and T. These designations have been selected to facilitate treatment and analysis of large groups of figures and have no organic or pathologic basis. Similar systems are described by Guild,[15] Fowler,[16] and Beasley,[8] but they employ many more types and grades of classification.

All audiometric curves falling within the range of plus or minus 10 db. from 1024 cps to 11,584 cps were classified as normal. Deviations at 256 cps and 512 cps were not considered in the typing since, under test conditions, these were the frequencies most readily masked by the variable ambient noise, and therefore subject to uncontrolled fluctuation. Threshold curves were empirically divided into the various types, viz., Type A—all cases showing no more than a 10 db. variation from the accepted zero-normal line in the frequency range 1024 cps to 11,584 cps; Type V—those cases showing typical and clear-cut V-notching or tonal dip (often referred to as the "airplane notch" or aviator's type of deafness); Type W—those curves showing the V-notch but having an additional loss of more than 10 db. in the higher or lower frequencies; Type T—those curves characterized by a progressive or variable high tone loss greater than 10 db.

In order to record the severity of the hearing loss, threshold curves were graded numerically, viz., Grade O—plus or minus 10 db. with reference to the zero-line; Grade 1—losses of 15-20 db.; Grade 2—losses of 25-40 db.; Grade 3—losses of 45-60 db.; and Grade 4—losses of greater than 60 db. It must be noted that this grading system takes into consideration losses at any one or more of seven discrete frequencies (1024-11,584 cps) and is not to be confused with the method of averaging losses for three or four selected frequencies.

In Chart I are demonstrated samples of the various types and grades of audiograms. Two grades are presented for each type, one designated by the circle and solid line, and the other by the triangle

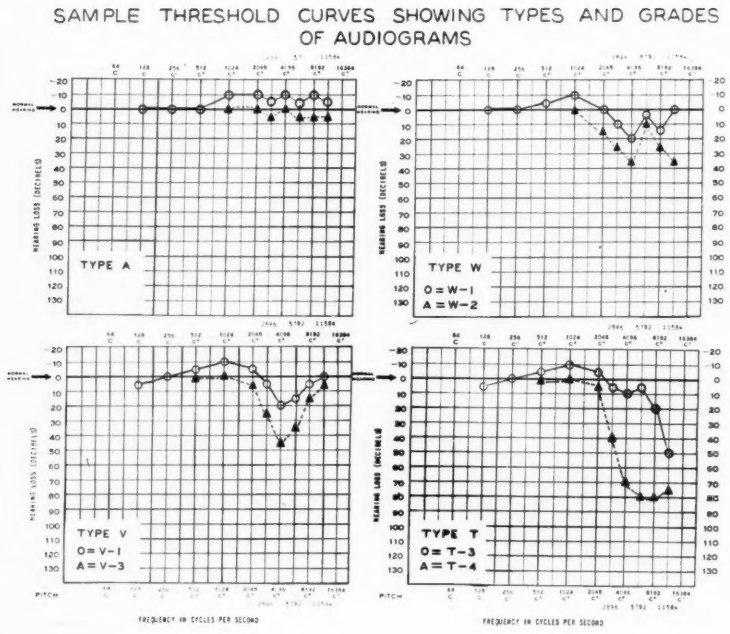


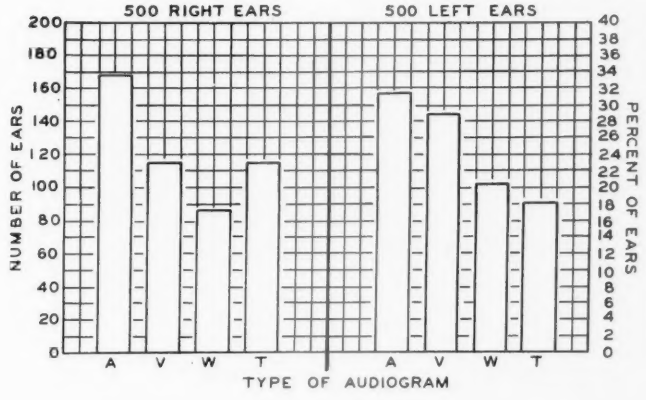
Chart I

and broken line. As an example, the curve illustrated by the triangle and broken line in the Type V sample shows a typical tonal dip with a maximum loss of 45 db. Since the greatest elevation of threshold at any frequency on this curve falls between 45 and 60 db., it is designated a Grade 3 hearing loss, and the audiogram classified V-3.

Each ear was tested separately without any planned order of testing right and left ears. A significant correlation between the hearing changes in the two ears of any given subject was found; hence, this series will not be considered as consisting of 1000 ears, but rather as 500 right and 500 left ears.

Results. Chart II shows the distribution of the various types of audiograms among the 500 subjects tested. It is seen that only 34 per cent of the right ears and 32 per cent of the left ears show threshold curves which do not deviate more than 10 db. below the accepted zero-line at any frequency from 1024 to 11,584 cps (Type

DISTRIBUTION OF 500 SUBJECTS ACCORDING TO TYPE OF AUDIOGRAM



	OBSERVED		EXPECTED*		OBSERVED		EXPECTED*	
A	170	34 %	165	33 %	159	32 %	165	33 %
V	118	23 %	132	27 %	147	29 %	132	27 %
W	94	19 %	99	20 %	103	21 %	99	20 %
T	118	24 %	105	20 %	91	18 %	105	20 %

*EXPECTED NUMBER AND PERCENTAGE OF THE FOUR TYPES OF AUDIOGRAMS ON THE BASIS OF EQUAL DISTRIBUTION BETWEEN RIGHT AND LEFT EARS.

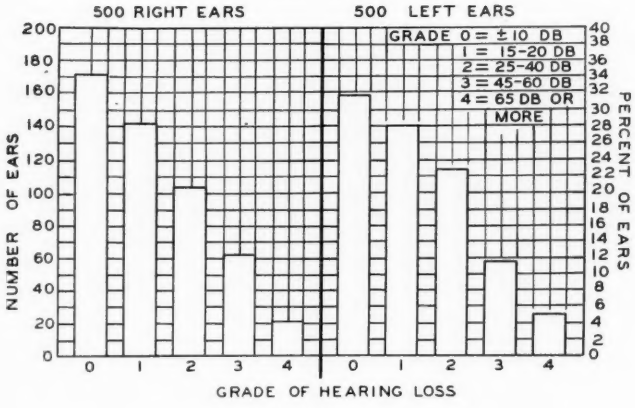
Chart II

A). This is in essential agreement with Crowe et al,[9] especially when it is recalled that their subjects are in a much younger age bracket.

Since it was possible for these normal (Type A) audiograms to vary 20 decibels (plus or minus 10 db. from the zero-line) at each frequency, it was of interest to determine the mean for each of the ten frequencies tested (128-11,584 cps). (Table 1).

With the exception of 128 and 2896 cps, all the means are within plus 3 db. of the zero-line. The large variations at 128 cps are the result of the masking effect of ambient noise filtering through into the sound isolated rooms. The changes at 2896 cps may be early evidence of the tendency toward V-notching in this vulnerable portion of the audiogram.

DISTRIBUTION OF 500 SUBJECTS ACCORDING TO GRADE OF HEARING LOSS



GRADE OF LOSS	OBSERVED		EXPECTED*		OBSERVED		EXPECTED*	
0	170	34%	164	33%	159	32%	164	33%
1	140	28%	140	28%	140	28%	140	28%
2	104	21%	110	22%	115	23%	110	22%
3	63	13%	61	12%	59	12%	61	12%
4	23	5%	25	5%	27	5%	25	5%

*EXPECTED NUMBER AND PERCENTAGE OF THE FIVE GRADES OF HEARING LOSS ON THE BASIS OF EQUAL DISTRIBUTION BETWEEN RIGHT AND LEFT EARS.

Chart III

TABLE 1.—AVERAGE THRESHOLD FOR TYPE A AUDIOGRAMS

(In Decibels) 170 Right Ears, 159 Left Ears

EAR	FREQUENCIES									
	128	256	512	1024	2048	2896	4096	5792	8192	11584
Right	6.3	2.4	2.0	0.9	1.4	4.6	2.9	2.0	1.0	2.1
Left	6.0	2.5	2.2	1.0	1.1	4.0	3.0	2.0	0.8	2.2

PERCENTAGE DISTRIBUTION OF HEARING LOSSES ACCORDING TO TYPE OF AUDIOGRAM

SHOWING ALL HEARING LOSSES OF 15 DB OR MORE AND FREQUENCIES AT WHICH LOSSES OCCURRED

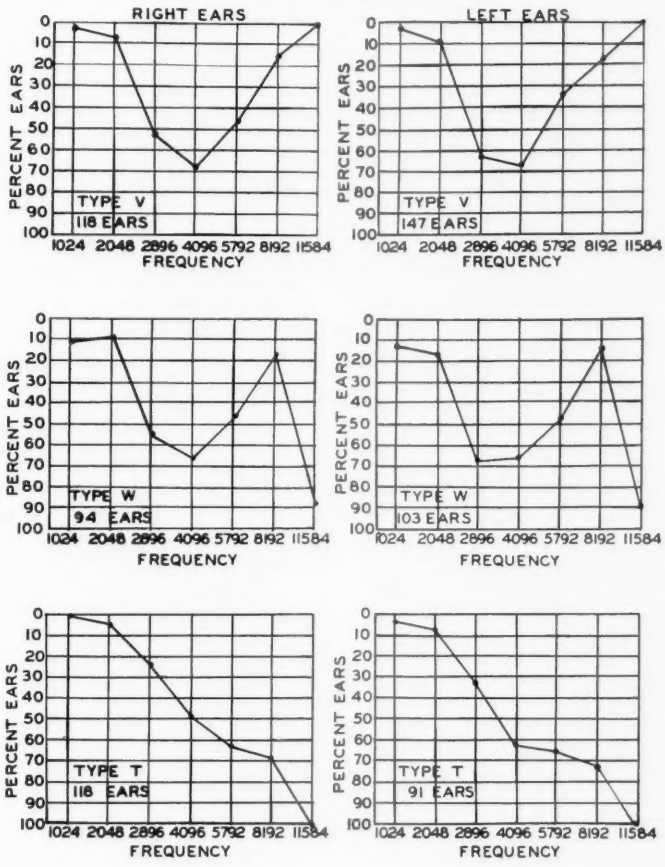


Chart IV

Tonal dips (Type V) occurred in 23 per cent of the right ears and 29 per cent of the left ears. This is a greater percentage than occurred in Loch's series of children[10] and confirms the impression that up to a certain undetermined age there is an increase in the occurrence of tonal dips. The higher incidence of this defect in the

left ear may be the result of greater exposure to noise and concussion during the use of firearms or its more frequent use for telephoning.

Type W audiograms occur in 19 per cent of the right ears and 21 per cent of the left ears and are considered the product of additional losses superimposed on the simple V-notch. Eighty per cent of the right and 77 per cent of the left ears designated as Type W show typical tonal dips of 15 db. or more.

The finding of Type T losses in so large a percentage of ears (24 per cent in the right and 18 per cent in the left) is consistent with observations among "normal" school children.[17] It is conceivable, but as yet unproven, that Type V hearing, under the influence of age and acoustic trauma, may be converted to Type W and, with continued abuse, into Type T deafness.

It is interesting to note the frequency with which the various types occur together (Table 2). Forty-nine and two-tenths per cent of the subjects showed bilateral similarity of hearing types. Of this number, 19.0 per cent were Type A; 11.0 per cent, Type V; 8.4 per cent, Type W; and 10.8 per cent, Type T. There appears to be a strong tendency for a given type of hearing in one ear to be associated with a similar type in the other.

TABLE 2.—Numerogram Comparing Bilateral Incidence of Types of Audiograms

		PERCENT LEFT EARS				
	TYPES	A	V	W	T	TOTAL
PERCENT RIGHT EARS	A	19.0	9.2	3.2	2.6	34.0
	V	6.6	11.0	4.0	2.0	23.6
	W	2.6	5.0	8.4	2.8	18.8
	T	3.6	4.2	5.0	10.8	23.6
	TOTAL	31.8	29.4	20.6	18.2	100.0

The distribution of grades of losses is shown in Chart III. A striking similarity and uniformity of the various grades of defects in the right and left ears is evident. There is a small percentage of severe hearing losses (Grade 4), and a preponderance of those showing normal (Grade 0) and minor hearing deficiencies (Grades 1 and 2).

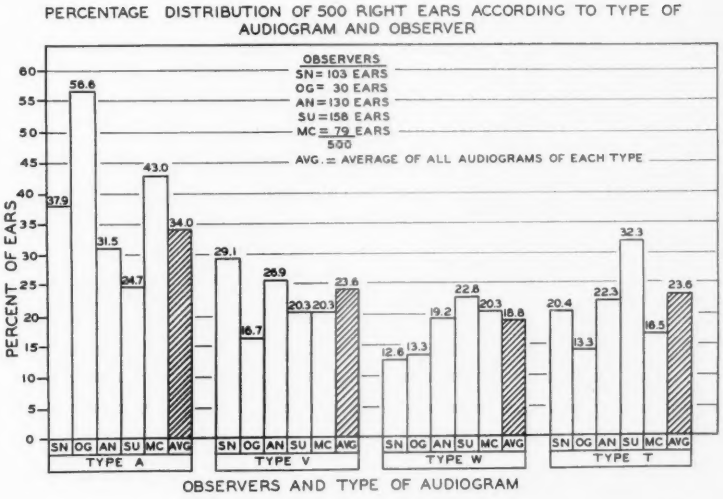


Chart V

Chart IV shows the threshold curves divided into types and charted as to frequencies at which losses occurred. The large majority of threshold elevations (losses) among the Type V curves occurred at 2896, 4096, and 5792 cps. Similar findings are evident in the Type W graph with the addition of a large percentage of losses at 11,584 cps. Among the Type T curves there is a progressive increase in the number of deficiencies of 15 db. or more at each of the higher frequencies.

Since the audiometric threshold curves were obtained by five different observers, it was considered desirable to analyze the entire data, for the purpose of comparing the types and grades of curves obtained by each examiner, with the averages of all the examiners. In Chart V are shown the percentages of each type of audiogram found by each observer and the average of each type of audiogram for all observers. It is seen that variations from the average do occur for SU, MC, and OG in the right ear, especially among Type A curves. Deviations are much smaller in the left ear (Chart VI). Care must be taken in comparing one observer with another because the number of cases examined is different for each observer. For example, the large variations from the normal of observer OG in Chart V are

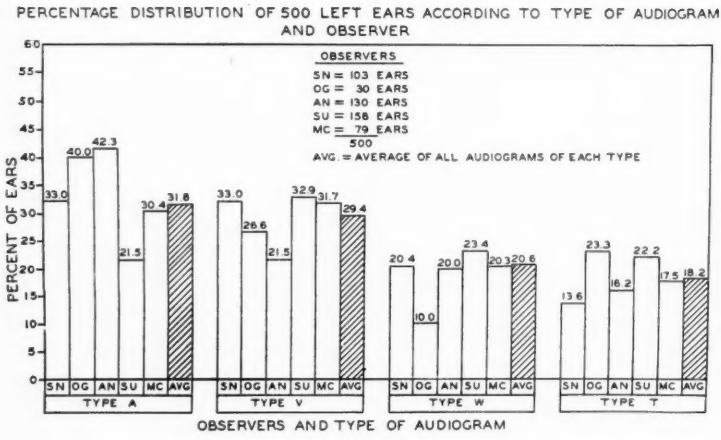


Chart VI

not as significant as those of SU since the latter examined five times as many subjects.

Differences are present which, in the right ear only, are statistically significant.* These are to be expected when several observers of somewhat different degrees of training are concerned in any large survey. Despite these differences in the results of the observers, the averages are still the best basis for making comparisons with other large bodies of data collected by multiple observers.

A re-evaluation of the tonal dip should be undertaken by those who would consider this type of defect pathognomonic of airplane noise exposure. It is essential to understand that the V-notch frequently occurs among children, young enlisted trainees, and animals, prior to any known prolonged acoustic fatigue.

The finding of such a small number of so-called Type A audiograms in this selected group of men emphasizes the important fact that there is a "normal" deviation from the audiometric zero-line threshold. This has been empirically accepted as plus or minus 10 db.,[8, 14, 18] but in this survey it has been shown that among other-

*These differences are statistically significant because as a set they may be expected to arise, by chance alone, less than once in one hundred trials on repeated sampling.

wise healthy 18- to 27-year-old men, significant deviations of the types and grades described in this report may occur, prior to any prolonged exposure to occupational or combat noise.

Since no large series of controlled precombat audiograms are available at the present time, it is suggested that this series of 500 audiograms be used as a control for field studies of postcombat cochlear damage.

SUMMARY

Auditory acuity tests were performed on 500 enlisted trainees between the ages of 18 and 27 years (mean age 21.1 years).

Thirty-four per cent of right ears and 32 per cent of left ears showed threshold curves which did not deviate more than 10 db. below the audiometric zero-line from 1024 to 11,584 cps.

Tonal dips of 15 db. or more occurred in 23 per cent of right ears and 29 per cent of left ears, prior to any recent exposure to aircraft noise.

Forty-nine and two-tenths per cent of the subjects showed bilateral similarity of hearing types.

Comparison of results of five examiners show some deviations but fairly uniform results are obtained.

This series may be utilized as a control for studies of postcombat cochlear damage.

Appreciation is expressed for the assistance of Mrs. Lynn Anderson in assembly and analysis of data; to Major Hubert B. Peugnet, MC, for calibration of audiometers; Captain Fred W. Ogden, MC, as observer; Lt. Earl L. Green, AC, and Lt. Arthur Kemp, AC, for statistical analysis; Mr. John J. Tucker for photography; and Sgts. John E. Sutherland and Roger N. McCown, as observers.

SCHOOL OF AVIATION MEDICINE.

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The preceding papers by former associates of Dr. Dean were contributed especially for this issue.

LXX

MÉNIÈRE'S SYMPTOM COMPLEX: OBSERVATIONS ON THE HEARING OF PATIENTS TREATED WITH HISTAMINE

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Ménière,[1] in 1861, first described the triad of symptoms vertigo, tinnitus and deafness, now generally known as Ménière's symptom complex. Since his description of the symptom complex appeared, many observations have been made in the literature. There is a striking lack of unanimity regarding the cause and treatment.

In Ménière's original report, pathological study revealed a bloody exudate in the semicircular canals, and the case reported was undoubtedly one of acute labyrinthitis. His pathologic observations, as we shall note, did not explain the clinical syndrome which he described.

Chronic catarrhal involvement of the middle ear has been advocated by many early observers[2-5] as being responsible for this condition. In 1920, Scott[6] suggested unilateral eustachian insufficiency or obstruction as the etiologic factor in 70 per cent of the cases.

Blake[7] suggested a fixation or ankylosis of the stapes with production of a loss of mobility of the transmitting apparatus of the middle ear and tension of the middle ear as the etiologic factor.

In 1883, Woakes[8] suggested increased intralabyrinthine tension as the cause of the symptoms. Cheatle,[9] in 1897, speculated on the possibility of increased tension of endolymph or perilymph as being the cause, as a result either of excessive secretion or obstruction to outflow. This condition has been referred to as "auricular glaucoma." Ferreri and Aboulker[10] and Portmann[11] also favored this

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idea. Hartsook,[12] in 1937, presented an excellent consideration of the possible cause. He also expressed the opinion that increased intralabyrinthine pressure was the cause of the symptoms.

Escat,[13] in 1906, suggested vascular spasm in the pathogenesis of Ménière's disease. In 1919, Lermoyez[14] stated that angiospasm of the vestibular or cochlear branches of the auditory artery could account for some instances of the symptom complex.

Dandy[15, 16] advanced the theory that the symptoms are similar to the paroxysmal attacks which one sees in trigeminal or glossopharyngeal neuralgia, and that they are the result of a primary lesion in the acoustic nerve itself. In 1934, Just[17] suggested the etiologic significance of angioneurosis of the acoustic nerve. Hughson,[18] in 1938, stated that a true toxic neuritis of the vestibular division of the cochlear nerve probably exists.

In 1923, Duke[19] reported instances of Ménière's syndrome which he attributed to allergy. The symptoms were relieved by epinephrine and the avoidance of certain foods. In 1931, Proetz[20] considered allergy of the middle and internal ear. He cited a case in which there was a definite sensitivity to milk and its products. Dean, Agar and Linton[21] also expressed the opinion that allergy to certain foods and drugs may produce the symptoms. Williams[22] said that Ménière's symptom complex may be regarded as an allergy "belonging to the larger syndrome of physical or intrinsic allergy as it involves the various structures of the head."

In 1940, Wright[23] advocated the removal of all possible foci of infection and said that the syndrome is secondary to infection elsewhere, usually in the nose, mouth or throat. Dean,[24] in 1941, and Shambaugh and Roberts,[25] in 1942, suggested, in addition to allergic surveys, a careful check for foci of infection.

In 1929, Dederding[26] advanced the theory of disturbed water metabolism as the etiologic factor. Mygind and Dederding[27-32] have studied the syndrome extensively from this standpoint. They expressed the opinion that there is an excess of fluid in the form of a generalized subcutaneous infiltration, an intracellular accumulation of fluid rather than an intercellular accumulation. They said that a nutritional disturbance produced by a capillary dysfunction results in deficient oxidation and an increased water binding efficiency in the cells, with subsequent intracellular edema. The same reaction occurs in the organ of Corti as the result of an increased permeability of the cells, probably caused by a deficient vascular, and particularly by deficient capillary, function in a predisposed region. They

stated that the deafness produced is of the conduction type. They noted an elevation of the lower limit of hearing and a lowering of the upper limit, and also a variability of hearing from day to day.

Furstenberg, Lashmet and Lathrop,[33] in 1934, advanced the theory that retention of electrolytes, chiefly sodium, and not the accumulation of water alone was the cause of the syndrome. Bartels[34] also agreed with this theory.

In 1938, Crowe[35] stated that "chemical, circulatory or pressure changes in the endolymph could produce the auditory symptoms by irritation or injury to the cells of the organ of Corti; and the vestibular symptoms by a cumulative effect on the organs of the vestibular nerve."

Hallpike and Cairns,[36] in 1938, reported the pathologic findings in two cases of Ménière's symptom complex; in 1939, Hallpike and Wright[37] added a third case. In all cases there was a marked dilatation of the endolymphatic system affecting chiefly the scala media of the cochlea and the saccule.

Lindsay,[38] in 1942, reported the micropathologic observations in a case of bilateral deafness in which there was evidence of greatly increased endolymphatic pressure. He said that such a case can be classified under Ménière's symptom complex. To him, increased endolymphatic pressure provides the most rational basis for the explanation of this symptom complex. He suggested that the production of the vertigo results from herniation of the utricular wall into the ampulla producing a distortion of the ampulla and thus interfering with the normal function of the cupula ampullaris. The auditory symptoms he explained on a physical or mechanical basis; that is, an interference in the transmission of sound in the cochlear fluid is the result of a distorted saccule, Reissner's membrane, and a disproportion of both the scala media and scala vestibuli.

Since 1938, the literature contains reports of 13 cases in which a histologic examination was made. Altmann and Fowler[39] reported the last three of these cases in 1943. In all of the cases, dilatation of the endolymphatic system was present, which indicated an increased endolymphatic pressure. No signs of inflammation were present in any of the cases. In all cases fibrosis of the perisaccular tissue was found and thought to be a predisposing etiologic factor.

In 1874, Jackson[40] noted the inability of a patient with Ménière's symptom complex to distinguish between A and C notes with the involved ear. Drury,[41] in 1929, stated that "the perception of mus-

ical notes is faulty in many of these cases." Shambaugh[42, 43] stated that diplacusis indicates a lesion in the cochlea which is the result of an alteration in the plane of vibration of the vibrating membrane. Since this is frequently found in Ménière's symptom complex he assumed that the lesion was in the labyrinth rather than in the nerve. He compared the cause and pathologic changes of this condition to those of iritis and called the condition "exudative labyrinthitis."

Sheldon and Horton[44] (1940) expressed the opinion that alteration in the permeability of the capillary wall with resulting local edema is the most likely etiologic factor. Whether the edema is intracellular or extracellular is not known. They said that it is not unlikely that a hemorrhage of blood serum alone, such as that comparable to the formation of cotton-wool exudate in the retina of the eye, may occur. Histamine was suggested as the possible agent affecting the capillary permeability.

Atkinson[45, 46] divided Ménière's symptom complex into two groups: (1) a vasodilator group in which there is a sensitivity to histamine, and (2) a vasoconstrictor group in which there is no sensitivity to histamine. The latter group is the larger. He stated that cases displaying characteristics of Ménière's syndrome may be placed in various groups according to the cause:

A. Lesions interfering with the function of the eighth cranial nerve.
   1. Lesions of the cerebellopontine angle.
   2. Degenerative vascular disease.

B. Lesions interfering with the function of the labyrinth:
   1. Alterations in intra-labyrinthine pressure
      (a) From without—stricture of eustachian tubes
      (b) From within—increased production of endolymph (Primary vasodilations; allergy; sensitivity to histamine.)
   2. Vascular disease
      (a) Angiospasm
      (b) Arteriosclerosis
   3. 'Toxic Labyrinthitis'
      Focal infection.

Other causes of Ménière's symptom complex suggested in the literature are epidemic meningitis (Gottstein[47]), lesions of the brain (Steiner[48]) and tumors (Sharkey[49]). Arterial causes, such as an arterial loop strangling or contacting the nerve, were reported by Dandy.

In 1912, Lake[50] suggested the following etiologic classification.

A. Peripheral causes
   a. Chronic progressive deafness of the middle ear
   b. Hemorrhage into the labyrinth and embolism
   c. Trauma

B. Vertigo caused by alterations in blood pressure
   a. Increased blood pressure
   b. Lowered blood pressure

C. General systemic causes
   a. Leukemia
   b. Gout (occasionally)
   c. Ocular symptoms
   d. Specific causes (syphilis)
   e. Cerebral anemia

Tobey,[51] in 1941, gave the following etiologic classification of vertigo.

A. Systemic diseases—endocrine disturbances, diabetes, avitaminosis and allergy
B. Toxic labyrinthitis
C. Mechanical disturbances—obstruction of the eustachian tube
D. Noninflammatory lesions of the middle ear
E. Apoplectiform conditions—hemorrhagic or traumatic
F. Unknown or obscure causes

Many reviews of the subject are to be found in the literature; those of Crowe,[35] Brunner,[52] and Simonton[53] are especially outstanding and complete.

Symptoms—Vertigo, tinnitus and deafness characterize Ménière's symptom complex. Any one of these symptoms may occur first. The vertigo is paroxysmal in nature and may occur any time of the day or night. It usually lasts approximately one to six hours. It frequently is described by the patient as a sense of whirling or rotation, or a sensation that the room is spinning around, or that objects in the field of vision are moving to one side or the other. It is almost always accompanied by nausea and vomiting. In many cases it is severe enough to cause the patient to fall, but only rarely does it produce unconsciousness. Many times it is followed by severe headache.

Tinnitus may be the first symptom noted. It may be paroxysmal or constant, and may vary in intensity and pitch. It may be unilateral or bilateral, and is usually more pronounced in the involved ear.

Noticeable loss of hearing may occur early or late in the disease, and is unilateral in most cases. In 182 cases reviewed, Walsh and Adson[54] reported unilateral involvement in 115 cases and bilateral involvement in 67 cases. Mygind and Dederding expressed the opinion that it is primarily a loss of conduction rather than a loss of perception. They noted a typical elevation of the lower tone limit and a lowering of the upper limit. Walsh and Adson reported that in 117 of 170 cases the loss of hearing was of a nerve type; in 11 cases there was a total loss of hearing; in 26 cases, there was a combined type deafness, and in 16 cases, there was a conduction type of loss of hearing (otosclerosis in one case and fixation of the stapes in five cases).

Treatment—Various types of treatment have been described in the literature. This is indicative that none have been too satisfactory in the past. Spontaneous remissions make it difficult to evaluate the effects of the treatment employed and may create false impressions of success.

Among the many drugs that have been used, one usually finds quinine (Charcot[55]), belladonna (Gowers[56]), hydrobromic acid, strychnine, valerian (Lake[50]), pilocarpine (Bishop[57]), potassium bromide, potassium iodide (McBride[58]), potassium nitrate (Walsh and Adson[54]) and potassium chloride (Talbott and Brown[59]).

Scott,[6] in 1920, suggested that treatment should be directed to the middle ear, and he advocated inflation of the eustachian tube. Atkinson,[60] in 1934, also suggested that treatment be carried out in this way.

Mygind and Dederding[28] (1938) expressed the opinion that disturbed water metabolism is the cause of this syndrome. They suggested the use of atropine in the acute attack. Their treatment consists in reducing the fluid intake, together with other general supportive measures such as outdoor exercise, proper rest, adequate caloric requirements, vitamins, calcium, ultraviolet light and physiotherapy. They reported that 81 per cent of the patients whom they had treated in this manner were permanently free of giddiness after three years or more. In 85 of 150 cases the hearing improved; in 47 cases, the hearing returned almost to normal, and in 24 cases (16 per cent) the hearing had not improved or had decreased. Eighty-three patients were followed for three years. In approximately half of the cases in which improvement in hearing occurred, the improvement was permanent; in 24 cases the hearing was better than it was

on admission but still decreased as compared to that at the time of dismissal, while in 8 cases the loss of hearing had progressed.

Furstenberg, Lashmet, and Lathrop[33] believed that the accumulation of water is not due to water alone but to electrolytes, chiefly sodium salts. They advocated that the intake of sodium be reduced to a minimum and that sodium also be prevented from accumulating in the body. They advised the hospitalization of the patients, the use of a carefully supervised diet free of sodium, and the administration of ammonium chloride. Successful results were obtained in 14 cases. Bartels[34] obtained good results by using Furstenberg's regime at the patient's home. Twelve of 18 patients treated in this manner obtained complete relief.

In 1940, Walsh and Adson[54] considered the relative merits of medical and surgical treatment. In a series of 182 cases reviewed, 115 patients had unilateral deafness and 67 had bilateral deafness. Complete deafness was shown not to indicate a guarantee of disappearance of vertigo as 11 patients were presented with complete deafness and vertigo was still present. In this series, 128 patients were treated according to Furstenberg's regime, 9 gm. of ammonium chloride was administered daily for three days and then the administration was discontinued for two days. Of these, 26 per cent reported improvement in the vertigo, 36 per cent reported complete disappearance of the vertigo, and 39 per cent said that the vertigo was unchanged. A modification of this regime also was carried out by substituting potassium nitrate for the ammonium chloride. It was noted that in 68 per cent of 94 cases of unilateral deafness the patients were improved. It was suggested that adequate medical treatment be tried before operation is employed.

In 1940, Talbott and Brown[59] studied the acid-base equilibrium of the blood and found no abnormal changes in cases of Ménière's symptom complex. They observed that sodium salts and soda bicarbonate could be administered orally or parenterally without precipitating an attack. In 48 cases they administered 6 to 10 gm. of potassium chloride a day and employed a low salt intake. Improvement in the vertigo was noted in all of these cases. In some cases hearing and tinnitus also improved. The precise action of the potassium chloride in these cases was not known. The diuresis produced by the potassium hastening the excretion of water and sodium chloride may have been a large factor.

In 1938, Alföldy[61] reported the use of nonspecific histamine therapy with good results. The histamine first was given intracu-

taneously and then subcutaneously in gradually increasing doses every two days. Ten to twenty injections were given. In 1940, Sheldon and Horton[44] first reported the intravenous use of histamine in the treatment of Ménière's symptom complex. In 1941, Horton[62] reported 49 cases in which this method of treatment was used. In 1940, Ingham[63] reported two cases in which histamine was administered intravenously with no recurrence of symptoms two and three months after the treatment was started.

In 1940, Harris and Moore[64] made an excellent review of Ménière's syndrome. As perceptive deafness associated with the vertigo is suggestive of a degenerative process and as many pellagrins have a rotatory vertigo, these investigators used nicotinic acid and thiamine in the treatment of this syndrome. In 17 of 20 cases, the patients became entirely free of vertigo and the remaining three were improved. Ten patients reported freedom from tinnitus, while the remaining reported it as being decreased. Twelve patients reported definite improvement in hearing while the hearing remained stationary in eight cases. It was found that two to three months of treatment was necessary before relief was noted. The treatment consisted of the administration of 250 mg. of nicotinic acid each day in five divided doses and of 10 mg. of thiamine twice a day. Audiograms, which were presented, showed improvement in hearing throughout the whole range of from 15 to 25 decibels, and in one case it amounted to 55 decibels at a frequency of 2,048 cycles per second.

Atkinson[45, 46, 65] divided cases of idiopathic Ménière's syndrome into two groups on the basis of the reaction of the patients to histamine. In the larger group of cases the patients are insensitive to histamine and the syndrome is the result of a primary vasoconstriction with a secondary vasodilatation. Intradermal tests with histamine, used in distinguishing these two groups, are carried out by using 0.0057 mg. of histamine base (0.10 mg. of histamine dihydrochloride). According to Atkinson a positive reaction is based on the appearance of pseudopodia. In cases in which the patients are sensitive to histamine, the patients are desensitized slowly. The intradermal test dose is repeated subcutaneously; every two to four days thereafter, the drug is administered subcutaneously in gradually increasing doses. When the maximal dose is reached it is repeated at intervals of four weeks. A second course has been suggested after six months, and possibly a third course. In patients in whom there is insensitivity to histamine, nicotinic acid is used because of its vasodilator effects. Usually an initial dose of 25 mg. is administered intramuscularly. This gradually is increased to the limit of tolerance

and is maintained at that level for approximately a month, then it is decreased gradually. The doses vary considerably, the extremes of variation are 10 mg. injected intramuscularly daily, 400 mg. injected intravenously, and 300 mg. administered orally. To some patients who were observed early, vitamin B_1 also was given in conjunction with the nicotinic acid.

Williams[22] expressed the opinion that division of idiopathic Ménière's symptom complex into two groups cannot be made on the basis of intradermal tests with histamine. We concur in the opinion expressed by Williams. He has not been able to confirm Atkinson's observations regarding the histamine skin tests. Williams believes that in cases of vertigo there is a gradation from "postural" types up through tinnitus and vertigo without deafness, to the classic Ménière's symptom complex. He has observed good results in cases in which histamine or nicotinic acid was administered regardless of the reaction of the patients to intradermal tests with histamine. We have made similar observations.

Many surgical measures have been advanced for the treatment of Ménière's symptom complex. Removal of the bones of the middle ear was suggested early. Burnett,[3] in 1899, suggested removal of the incus. Crockett,[66] in 1903, reported two cases in which removal of the stapes relieved the vertigo.

Many operative procedures on the labyrinth have been advocated. Total ablation of the labyrinth by means of injection of alcohol through the oval window was successfully employed by Wright[67] and Peacock[68] in 1938. Mollison[69] advised opening the horizontal canal and later also suggested the injection of absolute alcohol into the canal. Six cases in which both vestibules were ablated were reported by Gibson and Lake,[70] in 1908; total deafness, complete loss of equilibrium and complete relief of vertigo resulted. In 1911, Jenkins[71] reported a case in which a fenestration operation was performed; the opening was made only into the perilymphatic space of the horizontal canal. After the operation the hearing improved and the vertigo disappeared. In 1913 Page[72] reported a case in which a labyrinthectomy of the Hinsberg type resulted in a cure.

Day,[73] in 1943, reported the application of a coagulating current to the vestibule in cases of intractable hydrops of the labyrinth. A postauricular approach to the mastoid antrum was carried out. The horizontal canal was exposed, and just medial to the short process of the incus, a small opening was made into the canal with a motor

driven burr. A small needle was introduced through this opening toward the medial wall of the vestibule and a light coagulating current then was used.

Portmann[74, 75] referred to this symptom complex as "auricular glaucoma" and expressed the opinion that an increase in endolymphatic pressure on the endings of the vestibular and cochlear nerves produces a functional disturbance which brings about the development of symptoms found in Ménière's symptom complex. He advised surgical decompression by opening the saccus endolymphaticus.

Dandy[76-80] wrote many articles on the treatment of this symptom complex by intracranial division of the auditory nerve. He said that Charcot, in 1874, first suggested division of the auditory nerve to stop the symptoms. In 1902, Krause[81] suggested intracranial division of the auditory nerve for the relief of tinnitus, but not for vertigo. Parry[82] reported the first case in which division of the auditory nerve was performed. Facial paralysis resulted; the tinnitus was relieved but the vertigo continued. Frazier,[83] in 1912, followed the suggestion of Mills and performed an intracranial division of the eighth cranial nerve for aural vertigo but this procedure only partially relieved the vertigo. In 1928, Dandy[76] reported nine cases in which total section of the auditory nerve was carried out. None of the patients had subsequent attacks of vertigo. He expressed the opinion that the condition is primarily a lesion of the nerve and that the symptoms are similar to the paroxysmal attacks in cases of trigeminal and glossopharyngeal neuralgia. Thirty cases were reported in 1933 and 42 cases in 1934. In 1937, he discussed the advantages of partial section of the auditory nerve and said that a half to four-fifths of the cochlear portion of this nerve may be divided with practically no loss in hearing. In 50 per cent of the 401 cases reported in 1941,[80] the tinnitus disappeared after division of the nerve.

In 1933, Coleman and Lyerly[84] reported ten cases in which the auditory nerve was divided with good results. They expressed the opinion that the lesion is situated in the semicircular canals.

Walsh and Adson,[54] in analyzing the results of the surgical treatment of their series of 20 patients operated on at the clinic, reported the following results: Of the 13 patients who had complete unilateral section of the eighth nerve, nine had obtained complete relief of their vertigo, three had obtained great relief of their vertigo, and one had obtained no relief. Of the seven patients who had a subtotal section of the eighth nerve, four were completely relieved of vertigo, and three were greatly relieved.

A follow-up study indicated that 15 of the 20 patients were able to resume their regular work. Five complained that they were unable to resume their regular work.

Morgan and Baumgartner[85] obtained excellent results with cervical ganglionectomy. Resection of the stellate ganglion was cited by Biancalana,[86] in 1939, as a surgical procedure to be considered in Ménière's syndrome.

MATERIAL AND METHOD OF STUDY

This study is based on 25 cases of Ménière's symptom complex in which histamine was administered. Of the 25 patients, 18 were men and 7 were women (Table 1). The youngest patient was 27 years of age and the oldest was 65 years of age. The average age of all the patients was 46.4 years. The method of treatment, with the exception of minor modifications, followed that outlined by Horton,[62] in 1941. Two and seventy-five hundredths milligrams of histamine diphosphate (one milligram of histamine base) was administered intravenously in physiologic salt solution (250 cc.), a 5 per cent solution of dextrose (250 cc.), or in a 0.8 per cent solution of potassium chloride at the rate of 20 to 60 drops per minute. The blood pressure and the pulse rate were checked frequently during the injection. Histamine was administered daily in this manner for three to six days. In conjunction with or following the injections, subcutaneous administration of histamine (a 1:10,000 solution of histamine base) was begun; at first, the dose was 0.2 cc., but this was increased by 0.05 cc., twice daily, up to 1.0 cc. or until the optimal dose was reached. The patient was then instructed to continue with the daily injections of histamine and gradually taper off the dose according to the response to treatment. In several cases in which the symptoms were especially severe, intravenous injections were administered for many days. In many cases, histamine was administered intravenously during the subcutaneous "desensitization," to abort an attack of vertigo.

In this study we are primarily interested in the effects of histamine on the hearing. In this group of 25 cases, audiometric studies were made before and during treatment. A soundproof room was not available but the study was made under as uniform conditions as possible. All audiograms were made on the same audiometer and the tests were carried out by the same examiner. For definite improvement in hearing to be present it was decided that a change of at least 15 decibels in two or more frequencies was necessary. On the basis of + to ++++ the following classification was adopted:

+ = an improvement of 15 decibels in two or more frequencies

++ = an improvement of 15 to 20 decibels in two or more frequencies

+++ = an improvement of 20 to 25 decibels in two or more frequencies

++++ = an improvement of 25 to 30 (or more) decibels in two or more frequencies.

The presence of diplacusis was looked for throughout the whole frequency range in fifteen cases by comparing the given tone in each ear as to pitch.

Results—Audiometric studies revealed an improvement in the hearing in 12 of the 25 patients. In three cases only slight improvement was noted; in three cases moderate improvement occurred and in six cases the improvement was very noticeable. In the three cases in which only slight improvement in the hearing occurred, and in the three cases in which moderate improvement occurred, the histamine was given in physiologic salt solution. Of the six cases in which great improvement in hearing occurred, the histamine was given in physiologic salt solution in one, in 5 per cent glucose solution in one, and in an 0.8 per cent solution of potassium chloride in four (Table 2). In the twelve cases in which improvement occurred, three patients later returned to the clinic because of an increase in the deafness. All were given intravenous injections of histamine, but only one of the three showed an improvement in hearing. In eleven cases an elevation of the upper threshold of hearing was detected by the audiogram after treatment, while a lowering of the upper threshold was noted in three cases.

Diplacusis was found in 14 of 15 cases in which it was tested. It is interesting to note that in a large per cent of cases the tone was heard as a lower pitch in the involved ear.

Tinnitus was present in all but one case at the time treatment was begun. In ten cases no change occurred in the tinnitus. In two cases the tinnitus disappeared completely. It was greatly improved in eight cases and slightly improved in three cases. Questionable slight improvement occurred in one case. The frequency of the tinnitus ranged from 64 to 2,896 cycles per second; in most cases it was between 128 and 512 cycles.

Vertigo was the symptom which showed the best response to treatment. In 23 cases episodes of vertigo were occurring at the time

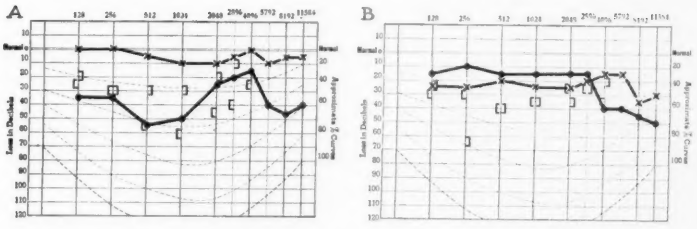

Fig. 1.—Audiograms in Case 4; A, before administration of histamine; B, after intravenous administration of histamine. Air conduction: right = . ; left = x. Bone conduction: right = [; left =] .

the patients came to the clinic. In nineteen cases there was a complete disappearance of vertigo while the patients were undergoing treatment and in two cases there was great improvement in this symptom. In two cases no change in the vertigo was noted.

Patients who received histamine frequently have noted a disappearance of fear and uncertainty, an improvement in the appetite and a subjective improvement in the hearing although audiometric studies did not reveal any change. It is possible that such subjective improvement in hearing is the result of the improvement in the general condition and sense of well-being. It demonstrates the fallacy of accepting the patient's word as to improvement of hearing, and also the necessity for checking the hearing with audiograms.

The symptoms recurred in several cases; in most instances, the symptoms recurred after an acute infection of the upper part of the respiratory tract.

The following three cases are illustrative of the results obtained in the series:

CASE 4.—A man, aged 28 years, had had a severe attack of vertigo three years before he came to the clinic. There had been no return of symptoms until three months before his visit to the clinic, at which time paroxysmal attacks of vertigo had been accompanied by nausea and vomiting. Tinnitus and decrease in hearing had been noted in the right ear two months before he came to the clinic. Histaminase had also been tried previous to his visit to the clinic but it had not produced significant improvement.

Eight intravenous injections of a 1:250,000 solution of histamine in a 5 per cent solution of dextrose were given on successive days. Subcutaneous injections of histamine administered twice daily were started, and given for eight additional days. The patient was instructed to continue to take nicotinic acid at home. At the time of his dismissal, the patient "felt much better". The vertigo had disappeared completely, the tinnitus was greatly reduced, and the hearing was im-

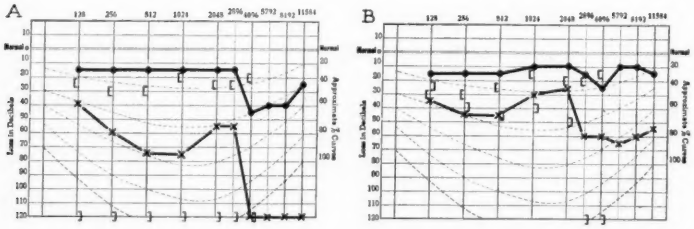

Fig. 2.—Audiograms in Case 7; A, before administration of histamine; B, after intravenous administration of histamine. Air conduction: right = . ; left = x. Bone conduction: right = [; left =] .

proved both subjectively and objectively. It is interesting to note the marked improvement in hearing in the right ear as recorded on the audiogram, and the moderate decrease in the left ear after treatment (Fig. 1).

Eleven months after the patient was dismissed and advised to continue treatment at home he wrote that he had been almost free of symptoms until two weeks previously, except for the "fear" of a possible attack. The hearing had varied from time to time.

CASE 7.—A man, 34 years of age, had had vertigo associated with nausea and vomiting, tinnitus and deafness on the left side, for three months. Headache had been present for a year.

Two intravenous injections of histamine in an 0.8 per cent solution of potassium chloride were given and subcutaneous injections of histamine were administered. The patient was advised to continue to take histamine at home. The headache and vertigo disappeared, and the tinnitus decreased in intensity. It is interesting to note that in this case no subjective change in the hearing was detected, but the audiometric studies revealed marked improvement throughout the speech range with an elevation of the upper threshold (Fig. 2).

CASE 22.—A man, aged 56 years, had had vertigo, bilateral tinnitus and deafness for a long time. The deafness was more pronounced on the left side. Histamine was administered intravenously in physiologic salt solution, next in a solution of dextrose, and later in an 0.8 per cent solution of potassium chloride. The patient was instructed to continue subcutaneous injections of histamine at home. We were very fortunate in being able to see this patient frequently from month to month. On this regime the patient noted a complete disappearance of the vertigo. The tinnitus and hearing varied markedly from time to time. The audiograms reveal the marked variation in both the right and the left ears which we noted in our studies (Fig. 3). Many other drugs also were tried without effect on the hearing and tinnitus.

SUMMARY

This paper is based on 25 cases of Ménière's symptom complex in which histamine was administered. Improvement in the deafness was noted in 12 cases; the greatest improvement was noted in the

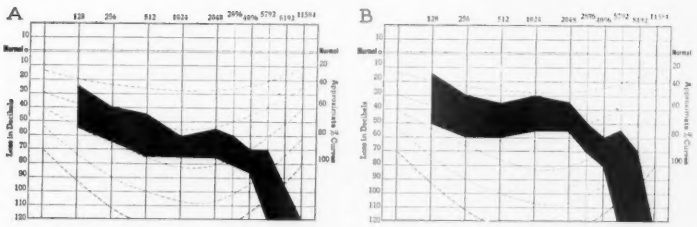

Fig. 3.—Audiograms in Case 22; A, variation in hearing (air conduction) in right ear; B, variation in hearing (air conduction) in left ear.

cases in which the histamine was administered intravenously in an 0.8 per cent solution of potassium chloride. Improvement in the tinnitus immediately following treatment was noted in 14 cases and improvement in vertigo was noted in 21 cases. It, therefore, may be concluded, although the number of cases studied is hardly sufficient to warrant drawing definite conclusions, that in a goodly number of cases of Ménière's symptom complex in which histamine is administered the hearing improves and there is considerable relief of tinnitus.

Misconceptions have arisen in the minds of the medical profession regarding intravenous histamine therapy. The method of giving histamine intravenously in a 1:250,000 dilution is a simple procedure, and at the clinic a large number of such injections have been given to patients with Ménière's symptom complex without producing any untoward effects.

MAYO CLINIC.

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TABLE 1

RESULTS OBTAINED WITH HISTAMINE IN TWENTY-FIVE CASES OF MENIERE'S SYMPTOM COMPLEX

CASE	AGE AND SEX		HEARING*	TINNITUS	VERTIGO	UPPER THRESHOLD OF HEARING	DIPLACUSIS
1	55	M	+ improvement	No change	None since treatment was started (5 mos.)	Not elevated	Present on right side
2	41	M	+ to ++ improvement	No change	Complete relief while under treatment	Elevated	Present
3	48	M	+ improvement	No change	Had been absent for 3 years	Elevated	Present
4	28	M	++++ improvement	Greatly reduced	Complete relief while under treatment at clinic	Not elevated	Not tested
5	43	M	++++ improvement at first visit	Completely gone on left; 75% improvement on right	Completely relieved while under treatment and for 6 months thereafter	Elevated	Present
			No change at return visit	No change	Much less	Not elevated	
6	46	F	++++ improvement	Marked improvement	None since treatment started (5 mos.) except mild dizziness on occasions	Elevated	Not tested
7	34	M	++++ improvement	Decreased	Complete relief while under treatment	Elevated	Present

TABLE 1—(*Continued*)

CASE	AGE AND SEX		HEARING*	TINNITUS	VERTIGO	UPPER THRESHOLD OF HEARING	DIPLACUSIS
8	59	M	++ improvement	No change	None since treatment started (4 mos.)	Elevated	Present
9	50	M	++ improvement	Slight improvement	Complete relief while under treatment at clinic. Only 2 mild attacks in following 2 years.	Lowered	Not tested
10	32	M	No change	No change	Complete relief while under treatment at clinic. No return of symptoms in 2½ years.	Elevated	Not tested
11	48	F	No change	No change	Complete relief while under treatment at clinic (7 wks.)	Elevated	Not present
12	27	F	No change	No change	No change	Not elevated	Present
13	51	M	++ improvement	Completely disappeared	Not present before treatment was started	Lowered	Not tested
14	46	F	No change	Slight improvement	No change	Elevated	Not tested
15	29	M	Further loss of hearing	Improvement of 15% to 20%	No vertigo while under treatment at clinic	Not elevated	Present

TABLE 1—(*Continued*)

CASE	AGE AND SEX		HEARING*	TINNITUS	VERTIGO	UPPER THRESHOLD OF HEARING	DIPLACUSIS
16	65	F	No change	Improvement of 85%	Improvement of 85% to 90%	Not elevated	Not tested
17	40	M	No change	Slight improvement (10%)	Not present before treatment was started	Not elevated	Present
18	63	F	No change	Decreased	No vertigo while under treatment at clinic. Still free of vertigo 6 weeks later	Not elevated	Not tested
19	54	M	No change	No change	Complete relief while under treatment at clinic	Lowered	Present
20	49	M	No change	Not present at time treatment was started	Complete relief while under treatment at clinic	Elevated	Present
21	54	M	No improvement	Improvement of 75%	Complete relief while under treatment at clinic	Elevated	Present
			Further loss of hearing; improvement of +++ to ++++ after treatment (return visit)	Improvement of 75%	9 months later no episodes of vertigo	Not elevated	Present

TABLE 1—(*Continued*)

CASE	AGE AND SEX	HEARING*	TINNITUS	VERTIGO	UPPER THRESHOLD OF HEARING	DIPLACUSIS
22	56 M	Variable; no improvement	Variable; no improvement	Complete relief since treatment started 2½ years before	Not elevated	Present
23	35 M	++ improvement	Much improvement	Much improvement while under treatment at clinic. Return of vertigo 2 weeks later	Not elevated	Present
24	45 F	No change	Completely disappeared while under treatment. Return of tinnitus 3 months later (first visit) No change (return visit)	Complete relief for 3 months after treatment started (first visit) No change (return visit)	Not elevated	Not present
25	55 M	No change		Complete relief while under treatment at clinic. Still free of vertigo 7 months later	Not elevated	Not tested

*The following symbols have been used to designate the various degrees of improvement in hearing:
+ designates an improvement of 15 decibels in two or more frequencies.
++ designates an improvement of 15 to 20 decibels in two or more frequencies.
+++ designates an improvement of 20 to 25 decibels in two or more frequencies.
++++ designates an improvement of 25 to 30 or more decibels in two or more frequencies.

TABLE 2

SOLVENTS USED IN CASES IN WHICH INTRAVENOUS ADMINISTRATION OF HISTAMINE PRODUCED IMPROVEMENT IN HEARING

SOLVENTS	IMPROVEMENT IN HEARING		
	SLIGHT	MODERATE	GREAT
0.8 per cent solution of potassium chloride	0	0	4
5 per cent solution of dextrose	0	0	1
0.9 per cent solution of sodium chloride	3	3	1
Total	3	3	6

LXXI

TINNITUS AURIUM: OBSERVATIONS ON ITS NATURE AND CONTROL

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What may be the nature and causation of tinnitus is a question the answer to which still eludes the puzzled otologist. One reason for this may be that the problem has been studied too exclusively from the purely otological angle, while the more general neurological outlook has been largely disregarded. Phylogenetically the auditory tract from end organ to sensorium is no other than a peripheral sensory tract modified to subserve the function of a special sense. It obeys the same general physiological rules as a peripheral nerve of common sensation and is subject to the same or equivalent disturbances of function, and for the same or similar reasons.

If the auditory tract is regarded in this light, the symptom of tinnitus becomes the homologue of paresthesia in the peripheral sensory apparatus and can be produced by precisely similar stimuli. Thus, just as paresthesia and the peripheral neuritis or neuropathy of which it is a symptom can be produced by external factors, mechanical and occupational, or by internal factors, such as the effect of chemicals and drugs, of infectious diseases and metabolic disturbances, so can tinnitus be produced by the same factors. The mechanical stimulus of loud noise (gunfire or riveting), the chemical intoxication of such a drug as quinine, the insult of infective diseases (influenza), of metabolic disturbances (diabetes), of vascular diseases (hypertension and arteriosclerosis), all these factors are as well recognized by the otologist as causes of tinnitus and impaired hearing as similar excitants are by the neurologist as causes of paresthesia and peripheral neuritis. And both recognize equally a large group of spontaneous or cryptogenic cases, the causative factor in which has still to be determined. It is with this last group that this paper is par-

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ticularly concerned—what may be its nature and how may it be controlled.

Cryptogenic tinnitus is the result of some abnormal stimulus, and is recognized in the sensorium as such. Though the subject likens his tinnitus for the purpose of description to sounds with which he is familiar, such as the roar of the sea, the hum of machinery, the hiss of steam escaping, yet he knows that it is not the same as any of these sounds. It has a different quality, often an unpleasant quality. Though like, it is yet different. It is, as Fowler[1] has aptly expressed it, an illusion of sound, and of a distorted and often a distressing sound. It is auditory paresthesia. This distortion of sensation is precisely parallel with that which arises in a damaged nerve of common sensation, which is "like" pins and needles or a constant burning, "like" but not the same.

Tinnitus, therefore, could be called equally well and with greater accuracy auditory paresthesia. This is merely to translate into pathological terms the physiological concept of Johannes Müller[2] of the specificity of nerve function, that stimulation by whatever means employed, electrical or chemical, of the nerves of any special sense organ produces a sensation appropriate to that organ, and to no other. "Sensation" said Müller, "consists in the communication to the sensorium, not of the quality or state of the external body, but of the condition of the nerves themselves, excited by the external cause." Similarly, pathological disturbance of the nerve of a special sense organ will produce symptoms, abnormal sensations, appropriate to that organ.

If this view is accepted of tinnitus as a paresthesia and the auditory tract as responding to disturbance of function in the same manner as other sensory tracts of which, because of their greater accessibility, we have more accurate knowledge, then at once many of the facts concerning tinnitus which have puzzled clinicians begin to have meaning and to fall into place. Thus, just as sensory paresthesia, if I may be allowed the tautological term for the purpose of differentiation, may vary all the way from a slight and short-lived sensation to a constant and even agonizing distress, so does auditory paresthesia or tinnitus vary from a passing hiss in the ear, which subsides as spontaneously and mysteriously as it came, to a constant surging roar of great intensity which renders life a burden. Just as in sensory disturbances division of nerves central to the lesion, and even amputation of a limb, may not relieve the patient because, by some mechanism which is not yet clear but which is gradually being clari-

fied, the original peripheral disturbance has come to involve pathways more centrally placed,[3] so with severe and long-standing tinnitus division of the cochlear nerve will often not relieve the patient of his noises. They remain to plague him, and presumably for the same reason.

Discussion of this parallelism could be extended much further, but I hope that enough has been said to make the point I wish, that tinnitus may reasonably be regarded as auditory paresthesia.

If the argument so far is well taken, then tinnitus is symptomatic of an active pathological disturbance in the auditory tract, and in the beginning at least in the infrasegmental division. If sustained, it is, as Fowler[1] has pointed out, a warning of impending deafness. Indeed, to discuss the symptom of tinnitus divorced from that of deafness is logically indefensible, since the two are inseparable; it may be excused only by limitations of space. For when there is tinnitus there is also impairment of hearing, even if only a temporary veiling during the short-lived bouts of tinnitus to which everybody is at times subject. Indeed, as tinnitus is the homologue of paresthesia, so is impaired hearing the homologue of hypesthesia, and total deafness of anesthesia. Again the parallel with sensory peripheral neuritis is exact. There we find associated with hyperesthesia or exaggerated response to stimulus a hypesthesia which is evidenced by a blunting of discrimination so that cotton and pin prick are no longer felt as such. In the auditory apparatus we call this combination tinnitus and deafness. As Fowler again has said, and I agree, these symptoms are the result not of a lesion in the cochlea but of a lesion in the nerve itself, of a neuronic disturbance probably of a biochemical nature conditioned by some local metabolic change. We are dealing in fact with a peripheral neuritis or neuropathy of the auditory nerve. Cochlear degeneration when it occurs (and it is by no means invariable[4]) is secondary to neurone degeneration—wallerian degeneration extending to the periphery.

If this concept of tinnitus and the associated impairment of hearing as evidence of an auditory neuropathy is acceptable, thus bringing the condition into line with peripheral neuritis or neuropathy in general, then the problems posed to the otologist by this syndrome are the same as those posed to the neurologist by the extremely difficult subject of spontaneous paresthesia. What is the process which brings about the neuronic disturbance? How can it be reversed? Why is it sometimes irreversible? What is the mechanism which determines the persistence of symptoms?

Difficult as these problems have proved to be to investigation even in so relatively accessible a region as the limbs, and largely unsolved as they still are, they become far more difficult in so inaccessible a region as that with which the otologist has to deal. In the limbs a frontal attack is usually possible. In the ear we are limited to the flank attack, partly because of a paucity of anatomical knowledge as regards the autonomic nerve supply, partly because of the difficulty of direct approach and the delicacy of the parts involved. The otologist is thus forced to an indirect approach. He must attempt by indirection to make direction plain.

The work of Leriche[5] and of many others has suggested that spontaneous sensory disturbances in the limbs are the result, at least in part, of a vascular disturbance, a vasospasm. Various procedures designed to produce local vasodilatation in the area involved have been adopted in the attempt to establish this thesis. Among these are injections of novocain around the sympathetic ganglia connected with the limb involved, periarterial sympathectomy, and division of sympathetic nerve trunks. Of these, the only one available for tinnitus, because of the inaccessibility of the region involved, is an attack upon the stellate ganglion through which pass all the sympathetic fibers to the head on the same side. It is, however, also possible by means of the appropriate drugs to produce general vasodilatation or general vasoconstriction, and observe the effect in lessening or increase of the tinnitus. Working on these lines, the following observations have been made.

Experimental Observations—All patients selected for these experiments had had troublesome or persistent tinnitus for months or years, thus lessening of the noise or even a temporary cessation, if it occurred, could not be ascribed to the spontaneous come and go of tinnitus which is a feature of many cases. All were tested with histamine intradermally by the technique already described by the writer[7] and the results were judged according to the criteria there laid down; all gave a normal response, hence were adjudged to have a primary vasoconstrictor mechanism.

1. Stellate Ganglion Block. A temporary block of the stellate ganglion by infiltration with a 1% solution of novocain has been carried out successfully, as shown by the production of a Horner's syndrome, on 18 patients, in some on more than one occasion. In all patients the tinnitus was of severe degree and had been continuously in existence for months or years. The results are shown in Table 1—no change in the tinnitus in 6 cases; a temporary increase in 2 cases, in one lasting as long as a week; in 10 cases improvement

lasting from 2 to 72 hours. Thus a definite change was effected in the degree of the tinnitus in 12 out of 18 patients, in 2 for the worse. This change for the worse is not necessarily evidence against the thesis, for similar deterioration has been observed in cases of pain in the limbs after the first sympathetic block, and yet repeated blocks have later proved successful. Unfortunately neither of these two patients would consent to further injections.

TABLE 1.—Stellate Ganglion Block: Novocain 1%

Effect on Tinnitus

TYPE OF DEAFNESS	IMPROVED	WORSE	NO CHANGE	TOTAL
Conductive	3	1	5	9
Nerve	5	0	0	5
Mixed	2	1	1	4
Total	10	2	6	18

One patient reported bilateral improvement, though less on the side which was not injected. This observation is of great interest. The same observation has been made in cases of causalgia. In this condition it is a well authenticated fact that pain may spread to the corresponding area of the opposite limb, what Livingston[3] calls the "mirror image" phenomenon. Here also measures to relieve symptoms on one side have resulted in bilateral relief. The same phenomenon was noted by Weir Mitchell,[6] though he did not use the term "mirror image." It is by no means uncommon in pain syndromes of long standing though the anatomical path of reference is not yet clear. This phenomenon may turn out to be of great significance in otology, where spread of deafness and tinnitus from the primarily affected ear to the contralateral ear at a later date is a usual occurrence, and where the audiogram in the more recently affected ear is often almost an exact counterpart or "mirror image" of that in the other.

One patient was subsequently injected with alcohol around the ganglion, first on one side and later on the other. As a result tinnitus, which had previously been of the severe type, was abolished entirely for three days on the injected side and then returned in much less severe degree; when the other side was blocked, tinnitus was markedly diminished so that it no longer awoke her at night. This improvement lasted about three months, but gradually the tinnitus

returned and became as loud as ever. Unfortunately on each occasion she experienced quite severe intercostal neuralgia, and because of this and the temporary nature of the relief, she refused further alcohol injection.

2. Vasodilator Drugs. The second available method, as far as the cochlear nerve is concerned, of producing a vasodilator effect is by means of drugs. A number of these have been experimented with, always in cases of severe and persistent tinnitus only. In order to provide a comparison with the results of stellate ganglion block, only the results of acute experiments are being considered at this point, that is to say, of the effect on the symptom of a single injection of a powerful vasodilator drug. The results are shown in Table 2. The effect of prolonged administration will be considered in the next section.

TABLE 2.—VASODILATOR DRUGS:

EFFECT ON TINNITUS OF A SINGLE INJECTION

DRUG	DOSE	NO. OF EXPERIMENTS	IMPROVED	WORSE	NO CHANGE
Acetylcholine	0.1 Gm. I. M.	14	8	1	5
Sodium Nitrite	0.1 Gm. I. V.	5	3	0	2
Prostigmin Methyl Sulfate	1:2000 Sol. 1 cc., I. M.	7	0	0	7
Magnesium Sulfate	10% Sol. 10 cc., I. V.	4	0	0	4
Strontium Chloride	10% Sol. 5 cc., I. V.	9	3	0	6

Although the effect of a single injection is not striking, as might be expected, nevertheless the results seem to provide some confirmation of those described above. Also as might be expected, acetylcholine gave the best results, though whether from its action as a vasodilator or whether by some more recondite action due to its function as a chemical transmitter of nerve impulses it is not possible to say. In all instances the effect was transient, though in one patient with unilateral tinnitus (acetylcholine) relief was complete for several hours.

3. Vasoconstrictor Drugs. If measures designed to provoke vasodilatation produce improvement, the contrary effect, deterioration, should result from vasoconstrictors. To determine this, a different procedure was adopted. Because tinnitus is apt to vary in its intensity at different times in the same patient, it was felt that conclusive evidence of change for the worse could only be obtained in patients who had previously been improved by other measures. The effect of vasoconstrictor drugs was investigated therefore only on such patients. The results have been striking in the chronic experiments, negative in the acute (Table 3).

TABLE 3.—Vasodilator Drugs

DRUG	DOSE	NO. OF EXPERIMENTS	IMPROVED	WORSE	NO CHANGE
Adrenalin	1:1000 Sol. 1 cc., I. M.	5	0	0	5
Ephedrine	Gr. 3/8 orally, t.i.d., several days	7	0	7	0
Amphetamine Sulfate (Benzedrine)	5 mg. orally, b.i.d., several days	5	0	5	0

Adrenalin, 1 cc. of 1:1000 solution, was injected intramuscularly in five patients. None of them reported any appreciable effect on their tinnitus.

Ephedrine, grains 3/8 in a capsule thrice daily, was administered to seven patients who had previously received a similar capsule containing sodium bicarbonate for a week with negative results. In four of these patients in whom tinnitus had been abolished or in whom it had become mild and intermittent, it became more intense and more persistent. In three patients whose tinnitus was mild, it became markedly more severe.

Amphetamine Sulfate, 5 mgs. twice daily, was administered to five patients who had received a similar tablet of sodium bicarbonate previously without effect. All complained of great increase of tinnitus within a week, two stopped taking it of their own accord after two days, and one patient with unilateral tinnitus asserted that it provoked tinnitus in the unaffected ear, an effect which has not been overcome even after many months.

So striking and consistent has been the bad effect of vasoconstrictor drugs in the 12 cases that I have sought since to avoid in patients complaining of tinnitus even the use of ephedrine nose drops.

The Control of Tinnitus. The observations described above suggest that the symptom of tinnitus may have its origin in a vascular disturbance involving the cochlear nerve. Because the same symptom in cases of Ménière's syndrome, which as I have attempted to show[7] also has a vascular basis, has been relieved or improved in a large proportion of cases in the vasospastic group by vasodilatation,[8] it has seemed reasonable to adopt the same method of therapy in similar cases not complicated by vertigo. For in my view, Ménière's syndrome is no other than chronic progressive deafness with tinnitus complicated by a vestibular disturbance. The two are fundamentally the same, etiologically speaking; the difference is only in the spread of the disturbance.

The patients here reported, therefore, have been treated on precisely the same lines as those I have already described for Ménière's syndrome,[9] using nicotinic acid as the vasodilator drug of choice. Other drugs have been tried but have been discarded. The effects of acetylcholine are very short-lived and it cannot be administered for the long periods necessary to obtain results without unpleasant side-effects. The same applies to sodium nitrite. Prostigmin has proved in my hands unreliable and at best of only temporary benefit. Histamine is useful in histamine-sensitive patients only, not in the majority of patients, who are histamine-insensitive. Drugs of the magnesium-strontium group are not potent enough.

But nicotinic acid—the acid, not the amide—is a powerful vasodilator. It can be given by all routes, intravenous, intramuscular and oral, and should be given by them all and in that order. It can be used over long periods of time without producing tolerance, and must be if satisfactory results are to be obtained. Moreover, clinically it appears to act with especial effect upon the vessels of the head. For these reasons I have used it almost exclusively, and the results shown in Table 4 have been obtained by its means alone.

The cases have not been selected; provided that patients complained of tinnitus and gave a normal response to histamine intradermally, all were grist to the mill. They have been separated, as a sop to convention, into two groups, according as to whether their accompanying deafness was predominantly conductive or perceptive as determined by the usual tuning-fork and audiometric tests. I have added in a separate category cases with vertigo, not in order to in-

crease numbers but for the reason stated above, that I hold that fundamentally there is no difference in mechanism whether vertigo be a symptom or not. Those who wish may discard this last group as not being valid.

TABLE 4.—Results In Treatment of Tinnitus Aurium With Nicotinic Acid, 1940-1943

Cases	Total Number of Cases	Number with Tinnitus	Results		
			Relieved	Improved	No Change
Deafness of middle ear type only	64	47 (73%)	10 (21%)	30 (64%)	7 (15%)
			40 = 85%		
Deafness of nerve type only	33	22 (67%)	3 (14%)	12 (55%)	7 (31%)
			15 = 69%		
Total (Deaf only)	97	69 (71%)	13 (19%)	42 (61%)	14 (20%)
			55 = 80%		
Deafness and vertigo (Ménière's Syndrome)	106	106 (100%)	13 (12%)	42 (40%)	51 (48%)
			55 = 52%		
Total, including Ménière's cases	203	175 (86%)	26 (15%)	84 (48%)	65 (37%)
			110 = 63%		

The results have been estimated conservatively. Relief means literally what it says, a complete absence of tinnitus, while improvement has had to be definitely asserted by the patient to qualify. Every effort has been made to avoid the pitfall of wishful thinking on the part of the patient, and the series is considered large enough to minimize the inevitable occasional error in this regard. It has been collected over the four years 1940-1943, and a number of these patients have been under continuous observation for more than two years.

It is interesting to note, particularly in view of the argument at the beginning of this paper and the rationale of treatment which has stemmed from it, that the greatest percentage of relief or improvement has been obtained in cases of conductive deafness (85% of 47 cases), whereas in cases of perceptive deafness only 69% of 22 cases have been relieved or improved. This is a finding which would scarcely have been anticipated and which cannot well be explained

along the lines of conventional otological thinking. In cases with vertigo the proportion is still less (52% of 106 cases), and this, I suggest, is because in them the disturbance is apt to be more severe (hence its spread) though not essentially different.

SUMMARY

1. An analogy has been drawn between tinnitus and the paresthesia of peripheral neuropathy and it has been suggested that tinnitus may be regarded as auditory paresthesia.

2. If this analogy is correct, then the problem facing the otologist in his attempts to relieve tinnitus is the same as that facing the neurologist in his attempts to relieve peripheral sensory disturbances, and the methods applicable to the one should be applicable to the other.

3. The cause of peripheral neuritis (neuropathy) is regarded by many as being on a vascular basis, at least in part, and experiments have been made and are described to determine the effect upon the tinnitus of altering the vascular supply to the cochlear nerve.

4. The results of vasodilator therapy upon a series of 175 patients complaining of tinnitus are given.

I wish to acknowledge the helpful criticism given by Dr. Samuel Brock in the preparation of this paper.

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LXXII

OTOLARYNGOLOGY ON THE HIGH SEAS

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Naval medical facilities have kept pace with the unprecedented naval expansion and world-wide warfare. For the first time in history hospital ships are adequate in number, superbly equipped and strategically located. When the blow fell at Pearl Harbor, the newest United States Navy hospital ship lay at anchor with the fleet. Within a few minutes after the first bombs fell, the boats of that ship were scurrying about the flaming, oil-covered waters, retrieving survivors and transporting them to the hospital ship. Prompt medical attention saved many lives. Some of those patients are again serving at naval guns.

A hospital ship on the fringes of a battle fleet exerts a profound influence on the personnel aboard the fighting ships. Her mere presence denotes adequate provision for the sick and injured. She is a potent factor in the maintenance of morale.

As the battered ships of the line limp into an isolated bay, many thousands of miles from home, the sleepless, weary medical officers and the wounded aboard can see no fairer sight than the trim lines, glistening white paint and the red cross of a hospital ship. Along the booms and gangways are whale boats, launches and gigs, with crews ready to cast off. Nurses in spotless, white, crisp uniforms line the weather decks. Within the ship, nurses, medical officers and corpsmen are at their stations for the reception of patients. By blinker and short wave they have already been informed of the number of patients to be expected, the predominating types of injury and those in need of immediate attention.

As the combat vessels anchor, the harbor is filled with the busy ambulance boats of the hospital ship whose activity never ceases until the last patient is aboard.

Read before the meeting of the American Laryngological, Rhinological and Otological Society, New York, N. Y., June 9, 10, 1944.

In the war in the Pacific, wounded men from distant battle fronts in dense jungle frequently reach a hospital ship or a mobile hospital within 8 to 24 hours. They may be flown a thousand or fifteen hundred miles in the large transport planes used for this purpose. The immediate treatment at advanced dressing stations is amazingly thorough and speed to the transport planes is ensured in spite of many difficult obstacles. Aboard the planes, medical attention is continued until an island landing field is reached where the casualties are transferred to mobile hospitals ashore or to a hospital ship in a nearby harbor. Waiting ambulances quickly transport patients to the nearest hospital or to the docks for the short trip to the hospital ship.

Whether the boats receive their casualties from ashore or from returning ships, they deliver their cargo to the quarterdeck of the hospital ship. As each patient arrives, an experienced medical officer designates the appropriate ward for treatment. In making this decision he is guided by tags affixed to the patient and notations concerning the patient received in preliminary messages.

Within a matter of a few minutes the injured have reached the ward where they are placed in comfortable bunks, with clean linen and soft pillows. Soap and water is profusely used, for cleanliness is imperative. The mere removal of oil, dirt and powder not only improves the immediate appearance of the patients but buoys morale. Bandages are removed and replaced as necessary, casts and splints rearranged, plasma or blood administered and sedatives given.

The compactness of the arrangements on a hospital ship make it a simple matter to transfer patients to the x-ray department or operating rooms. An abundance of trained personnel expedites such transfer.

Cleanliness, quiet, the sense of safety and the general atmosphere of efficiency have an immediate effect. For the first time in months, soldiers, marines or sailors may be experiencing a clean comfortable environment. Some of the intangibles are as effectual as medical and surgical measures. The therapeutic effects of ice cream, cold fresh milk and fruit juices are amazing. After 24 hours, the strained, hopeless, dazed faces begin to smile and grin. Wisecracks and joshing fill the ward. They are on their way back to health.

Four to six hundred wounded may be taken aboard a hospital ship within several hours. Then, "Station all special sea details", over the loud speakers will indicate that in a few minutes the anchor will come up and the ship be under way.

In the otolaryngologist's wards, many of his patients have already been examined and treatment started, but until port is reached he will sleep little and rest not at all. Those assigned to him have injuries which are in the upper respiratory tract, ears, eyes or skull. The ophthalmologist assumes charge of his particular group. In addition, almost every patient will present a number of injuries to other parts of the body. Occasionally, these other injuries may prove to be of more moment and then prompt transfer to the care of the medical officer best equipped to deal with that injury is made.

It is necessary for the otolaryngologist to assume temporary responsibility for the treatment of major wounds, burns and shock until such time as his colleagues can relieve him of that responsibility. Some casualties may have innumerable shrapnel wounds and many foreign bodies. The entire body must be searched for evidences of injury. The recognition of cessation of circulation in a leg because of damage to the popliteal artery, if promptly made, and immediate attention directed to the injury, may prevent amputation later on. The neurosurgeon, orthopedic surgeon and others must be promptly called into action where they are needed. It is the team work between the medical officers of our hospital ships that has made possible the amazing record in the treatment of casualties during this war.

When the otolaryngologist regards his own domain under reasonable control, he tours the other wards, examining and treating those to whom their medical officers direct him, and in a swift survey attempts to pick out injuries in his own field that deserve attention but which may have been overlooked by physicians primarily concerned with other types of injury.

To give proper attention to the casualties, many hours may be spent in the operating room. Surgery becomes as simple to perform at sea as ashore. Only during severe storms of hurricane intensity is there need to delay operations because the ship is under way. There is need for much immediate surgery and for many reasons other than life-saving measures. Early repair often saves months of convalescence and the need for extensive plastic surgery. Even bronchoscopy is possible at sea and is frequently essential for the removal of foreign bodies in the lower respiratory tract as well as for diagnosis and assistance to the chest surgeon. Every naval engagement and every land battle will provide new types of injury which will tax the ingenuity of the otolaryngologist to the utmost. In many instances he may improvise entirely new techniques to manage unusual problems. Because of his strategic position and excellent

equipment he carries a grave responsibility. Here is an opportunity to record priceless data for the guidance of other medical officers. His observation of wounds and injuries comes at a time when conclusions regarding the effectiveness of different forms of therapy can be clearly made. If observant, and meticulous in his observations, he may be able to record facts of inestimable value to medicine in general and to his specialty in particular.

Professonal activity is not the sole cause for loss of sleep. The medical officer stands at his station for an hour before sunrise and for an hour before sunset. Since his station is with his patients, he readily acquires an intimate knowledge of their whims and fancies. Unconsciously he develops a feeling of responsibility for them which transcends that of most other physician-patient contacts. During these dangerous hours when he is prepared for sudden contingencies, he assures himself that every man has his life jacket properly adjusted, that the corpsmen know what to do at abandon ship, and that nothing is amiss in administration of therapy. During the day he may interrupt his schedule to attend boat drills during which he supervises the crew of his life boat or life raft.

Patients are treated as if they were to remain aboard until ready for active duty. Actually they are transferred either at sea to another ship or upon reaching port to a hospital ashore. In either case the respective medical staffs are on the alert for the reception of the casualties. Even at night and at sea the entire load of casualties may be transferred within a few hours. Again this is possible only through the training and organization of the personnel aboard the ships.

If the hospital ship finally slips into dock in a port far from the dangers of the combat zone, hospital trains or long lines of ambulances are awaiting at the dock. Here the transfer of the patients may occupy two or three hours. Usually trained litter bearers from shore organizations expedite the transfer and within a few hours the wards of the ship are again empty.

The activity of a hospital ship is not altogether dramatic. One of its most important functions is to provide medical and surgical attention under all circumstances. Between the episodes of combat there are relatively long intervals of weeks or months when the hospital ship lies at anchor with a fleet or task force. During this time the combat vessels are busy with preparations, reloading and refueling, making repairs and engaging in practice. Among the many thousands of men manning these ships, about the same percentage

of disease and injury will occur as in a comparable group in civil life. The hospital ship then assumes all the functions of a large civilian hospital, including investigative and therapeutic activities, consultation service for other ships, and research. While there are adequate medical personnel aboard all ships, the personnel and facilities for highly specialized services can only be provided and concentrated aboard a hospital ship.

The otolaryngologist aboard a hospital ship is enabled to practice his specialty under ideal conditions. He has constant control and supervision of his patients. In fact he is never more than a few minutes from them at any time, day or night. If he desires consultation with his colleagues, they are available within a few minutes for they may be reached by an automatic dial telephone system. There is no delay in securing x-ray studies or laboratory cooperation. He is assisted by nurses and corpsmen who have been especially trained and are experienced in the management of otolaryngological problems. Above all they are adaptable to the sudden changes from routine otolaryngological practice to the reception of a flood of casualties.

The Eye, Ear, Nose and Throat Department is compact. Its wards, clinic and operating rooms occupy contiguous spaces. There is no lost motion and no fatiguing journeys through endless hospital corridors. Floor or deck space six feet by six feet will provide a room large enought to contain a hydraulic adjustable chair for Bárány examination, a Ferree-Rand perimeter, a tangent curtain, an audiometer and a transillumination outfit. In addition, the room may be used for ophthalmoscopic examinations. The treatment cubicles contain adjustable hydraulic chairs, treatment cabinets, suction, pressure and electrical outlets, instruments and medicaments, fountain cuspidors and sterilizers. It would be difficult to find an equally complete group of treatment units in civilian hospitals or in the offices of civilian physicians. The provision for electrical outlets and lighting satisfies the most critical.

While attached to a fleet lying at anchor, a hospital ship is an exceedingly busy organization. Patients are received not only from the nearby ships but from units and hospitals ashore. At such times, an Eye, Ear, Nose and Throat Department, with one ophthalmologist and one otolaryngologist, may average from 1800 to 2300 patient visits a month. Tonsillectomies, submucous resections and ear operations approximate the number that would be gleaned from a similar service in civilian hospitals. For some reason, men aboard ships are prone to frequent recurrent throat infections. These are

incapacitating for several days during each attack and the time lost in the sick bay may affect the efficiency of the ship's organization. Following tonsillectomy, the elimination of the time formerly lost in the sick bay frequently is a considerable factor in improving the efficiency of the ship's personnel. Free nasal respiration is important in divers, submarine and flying personnel. Therefore submucous resection or other intranasal surgery assumes importance in keeping trained personnel in action when they might otherwise be relegated to an inactive role.

In the tropics, mycosis of the external auditory canal is not only common but it assumes proportions and severity which impair the military effectiveness and efficiency of an entire unit. In controlling infections of this sort the otolaryngologist will need the utmost in ingenuity, persistence and imagination. His efforts, however, will often be rewarded by the knowledge that he has contributed to the success of an important action by keeping key men at their posts in time of emergency.

Rare lesions due to tropical diseases will be referred by medical officers on other ships or from shore hospitals. There will be variety in clinical material. Individuals who have never sneezed or wheezed or itched will develop allergic manifestations from tropical vegetation which the botany books ignore. If his bump of curiosity is developed, he may investigate those unexplored diseases provided by the neglected natives in isolated ports.

A hospital ship also serves as a postgraduate school afloat. Aside from the medical library, which is usually an excellent one stocked with current periodicals and the latest textbooks, the medical meetings and clinico-pathological conferences offer a continuous opportunity to keep abreast of recent trends and developments. Meetings are attended by medical officers of other ships and shore stations as well as by the staff. They thus become a clearing house for ideas and permit interchange of experience. Far reaching changes in policy and treatment are often initiated here. The contacts with able teachers are stimulating and the medical staff of a hospital ship are in a position to foster a program which is an acceptable substitute for postgraduate instruction.

All is not work at sea. The journeys back to the combat zones may be pleasantly occupied by rest on deck when the sea and sky display their ever changing repertory of color and motion. Physical well-being can be maintained by deck sports and games. Social contacts with fellow officers are pleasant interludes.

In any port there is much to amuse as well as educate an interested spectator. Native villages, tropical vegetation and military installations are of endless interest. The visit to a leper colony or a native hospital will be long remembered. Above all there will be new friends to make and occasionally an old friendship to renew. At sea or ashore, the otolaryngologist of a hospital ship will rarely find time hanging heavy on his hands.

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LXXIII

BIPOLAR TONSILLECTOMY

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Pharyngologists are familiar with the appearance of children's tonsils when removed by the automatic instrument. The lower pole has the same smooth-appearing capsule as has the upper pole. The question arises as to why the adult tonsil, when removed by dissection, often has a rough appearing surface of the capsule of the lower pole, while the upper pole capsule is smooth and white and closely resembles that of the child. Furthermore, one wonders whether or not the lower pole could not be dissected as easily as the upper pole if a proper method of approach were found. We have tried to find this approach.

The work of Fowler[1] and the dissection of Beecher and Todd,[1] who described the tonsillopharyngeus muscle, were of great assistance. These authors state that the tonsillar bed appears different with the tonsil in situ from what it does after removal. The tonsillopharyngeus muscle has a broad origin from the palatopharyngeus muscle. The upper fibers curve downwards and medially to be inserted into the equator of the tonsil, while the lower fibers curve upwards and medially to be inserted into the same equator. This is the only muscle of the pharynx which actually enters the capsule. Dissection in the tonsillar fossa from above downwards goes well until the tonsillopharyngeus muscle is reached. The lower fibers of this muscle carry the dissecting instrument down and outward away from the field and into the palatopharyngeus muscle.

For this reason the dissection is stopped when the equator is reached. This lies approximately at the junction of the middle and lower thirds of the tonsil.

One recognizes that he has gone far enough when he encounters the resistance of the enveloping fibrous sheath of the tonsillopharyngeus muscle. The dissection is then started from below.

Complete description of the method follows: Preanesthetic medication is given according to the method of Cullen.[2] Pentobarbital sodium (nembutal) gr. 1½ is given by mouth 45 minutes before

operation. Morphine sulfate gr. 1/6 to 1/4 and scopolamine hydrobromide gr. 1/150 to 1/100, diluted in 5 cc. of physiological sodium chloride solution are injected intravenously 15 minutes before the operation. The dosage of morphine is the same whether given subcutaneously, intramuscularly or intravenously. The time of optimum effect alone varies. The ampule form of scopolamine is preferred to the tablets as the latter deteriorate with age and may become toxic.[2]

If the patient comes into the hospital early enough, the nembutal is given two hours before the tonsillectomy and the morphine and scopolamine mixture injected subcutaneously one and one-half hours before. Another schedule is: nembutal one and one-fourth hours before surgery and morphine and scopolamine intramuscularly forty-five minutes before.

Routine preanesthetic medication is avoided and patients are individualized according to Cullen's method.[2] Age is the prime factor in deciding upon the dose. Older people require less due to the fact that their metabolic activity becomes reduced. Active, young, healthy adults require most. These receive morphine gr. 1/4 and scopolamine gr. 1/100. The ratio of 25 to 1 is maintained in dosage for those of other age groups. People over 50 receive morphine gr. 1/6 and scopolamine gr. 1/150. Obese and hypothyroid types of individuals receive proportionately less as they need less and will not tolerate the drug as well. Wiry, alert, apprehensive types require more.

The advantage is that we have a placid, nonapprehensive patient at the time of operation. The pharyngeal mucosa is anesthetized and the gag reflex largely abolished. Some patients have no memory of the surgical procedure, even though they had been cooperative at the time. Infiltration technique is used rather that general anesthesia: 1% procaine in physiological sodium chloride solution, with eight drops of adrenalin to 20 cc. of the solution, is used. Less than 20 cc. are ordinarily needed.

A partial ring of infiltration is made along the margins of the anterior and posterior pillars by separate injections. The first is made into the anterior pillar below and lateral to its insertion into the base of the tongue. The depth to which the needle is carried is just below the surface of the mucosa. A second injection is made at the insertion just described, and another higher up on the anterior pillar. The plica semilunaris is next injected and one or two spots are se-

lected for injection on the posterior pillar. Thus the whole margin of the fossa is infiltrated, with the exception of the base.

Deep infiltration is secured by use of a curved tonsil needle and by going mediolaterally behind the tonsil and close to its pseudo-capsule* at the tonsillar equator. A second injection may be made lower down, but no other deep infiltration seems to be required.

Incision of the mucosa is made before the tonsil-seizing forceps are applied. This is carried from near the lower end of the anterior pillar upwards along the free margin of the anterior pillar, back and around the edge of the plica semilunaris and down the posterior pillar. The opposite tonsil incision is next made in the same manner. Care is taken to incise only through the mucosa.

Now the tonsil is grasped and the Marschik dissector is used to expose the pseudo-capsule of the upper pole. Care must be exercised to get down to the pseudo-capsule which is ordinarily smooth, white, and glistening. Generally speaking it may be said that if one does not see this silvery barrier but instead sees a red, muscular-appearing field he is not down to the capsule. It might also be pointed out that if one finds oneself working in a red, bloody, soggy mass of tissue, he has already entered the parenchyma of the tonsil itself. He must now clear the field of blood by use of sponges or suction until it is sufficiently dry to permit clear visibility. Then one may search above, sometimes far above, until he can identify and grasp the tough, thin fibrous sheet forming the pseudo-capsule and proceed to dissect along its dorsolateral surface.

An Allis clamp with a long handle is used to grasp the capsule above in order that added traction may be secured. After sweeping over the dome of the upper lobe the scissors are used to dissect the tonsil from the fossa. These should be applied very close to the pseudo-capsule. The slightly opened scissors may be used with a pushing motion to expose individual groups of fibers. These groups of fibers may then be excised.

When dissecion of the upper pole is completed the Allis clamp is removed. The incision of the mucosa of the anterior pillar is now carried down to, or near, the attachment of the pillar to the base of the tongue.

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*The capsule of the tonsil is only a part of the fibrous sheath covering that organ. Separation of this sheath by dissection results in two fibrous layers.[3] The one which covers the tonsil is known as the capsule. The other covers the tonsillar fossa. As the sheath covering the tonsil is not a true capsule, it will hereafter be referred to as the pseudo-capsule.

The Allis clamp is now used to grasp the anterior portion of the pseudo-capsule of the inferior pole. This is really the fibrous layer of the plica triangularis.[4]

The inferior pole is treated in much the same manner as the upper. A Marschik dissector is used to expose the smooth, white capsule. There is a slight variation in the problem here, due to the presence of the plica triangularis. After the dissection of the lower pole has been well started, the mucous membrane layer of this plica becomes visible. Its thin, cut anterior edge lies free, as it has become elevated from the pseudo-capsule by dissection of the lower lobe. As it often contains lymphoid tissue, it is removed. In the case of large tonsils, the plica triangularis is attached directly to the tongue. When the tonsil is smaller, the plica is reflected directly onto the lateral pharyngeal wall. In either case the mucous membrane layer of the plica is severed at its base by the cut of the scissors. In cases in which the plica triangularis is attached to the base of the tongue, this method provides an accurate separation of the tonsil from the tongue under direct vision. One may now comfortably complete dissection of the inferior pole.

It is felt that long continued postoperative distress in this region is caused by the dissection being erroneously made deep into the palatopharyngeus muscle. The resulting cicatricial tissue binds the tongue to the lateral wall of the pharynx and thereby interferes with the normal mobility of the tongue.

The tonsillopharyngeus muscle has now been isolated both above and below. One may complete the removal of the tonsil by application of the snare. It is hoped that sharp dissection may be used here also. However, so far the snare has worked better for the final severance of the tonsillopharyngeus muscle.

Bleeding is controlled by first grasping the severed end of the bleeding vessel with the hemostat. For suturing, No. 00 plain gut, 30 inches long, fastened to the end of a pliable, curved, eyeless cutting needle is used. It is felt that the eyeless needle is superior for the reason that there is less trauma produced. What is more important for the immediate situation is that less bleeding is caused. The needle is bent to conform to the requirements imposed by the shape of the fossa and shallowly inserted through the fascia and muscle above the bleeder in a lateromedial direction. Then it is inserted below the bleeder in the same direction. The hemostat is now removed. The pattern in which the gut lies is that of a capital "Z." A square knot is tied, as follows: An end of the gut is grasped in each hand. The

gut is allowed to run over the tips of the index fingers. Then it is thrust into the throat in order that tension on the gut may be near the site of the bleeder when the knot is tied. This is a similar method to that used in knot tying in abdominal surgery. It will be seen that the knot brings the four corners together. These corners represent the entrance and emergence of the needle above and below. The vessel is now securely tied, no pedicle being left in the throat.

SUMMARY

A method of tonsillectomy is presented which is governed in a large measure by the peculiar anatomy of the tonsillopharyngeus muscle. It is hoped that this description will be of some benefit to those who remove tonsils surgically.

72 West Second St.

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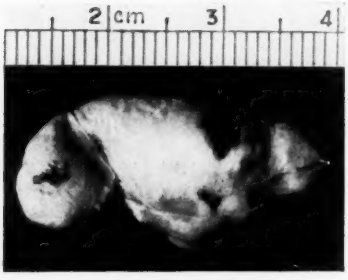
Clinical Notes



Photograph of tumor coughed up.

LXXIV

FIBROSARCOMA OF THE LARYNX

JOHN H. FOSTER, M.D.

HOUSTON, TEXAS

H. M. A., age 31, was referred to me December 9, 1943, on account of a laryngeal tumor with considerable dyspnea. The patient, who had been discharged from the Army on November 23, 1943, was the son of a physician in Dallas and had taken one year in medicine before enlisting.

On July 3, 1943, the patient coughed up a firm piece of tissue about the size of an English pea. This was followed by some hemorrhage that lasted for 30 minutes. The only symptom noticed prior to this was a slight râle on inhaling. About ten days later the patient coughed up another piece of tissue slightly larger than the first. Again bleeding occurred for half an hour.

On July 27, after suffering dyspnea for three days on lying down the patient coughed hard and brought up a well-defined tumor about

Read before the Sixty-Sixth Annual Meeting of the American Laryngological Association, New York, N. Y., June 8, 1944.

the size of an almond. He was hospitalized and examined by the indirect method, but nothing was found. He was then sent to a large General Hospital where a direct examination and an x-ray examination showed the site of the tumor to be subglottic.

On August 10 and August 23, attempts at removal were made through a direct laryngoscope and on September 3, another laryngoscopy was done and the larynx was reported free of growth; but again on October 11, another operation was done and some tissue removed. The patient was discharged, as above stated, on November 23 and was apparently well except for coughing up dried, greenish crusts occasionally. He thinks he caught cold on the way home and had a slight hemoptysis. Breathing gradually became obstructed until he was referred to me.

Examination by indirect laryngoscopy revealed a subglottic tumor that obstructed the airway to such an extent that there was considerable dyspnea. It seemed to be attached anteriorly. The examination was otherwise negative. An x-ray examination was made with the following report:

"There is a tumor measuring 2 cm. in diameter arising from broad base on the anterior portion of the trachea just distal to the larynx with an elevation of 1.5 cm. extending into lumen of the trachea. The tumor mass is sharply-circumscribed, having a somewhat irregular summit, containing no calcifications. The lumen of the trachea is considerably narrowed.

"The lungs show normal aeration, are free from infiltration or consolidation. The heart and the mediastinal structures are within normal limits; the bony thorax and the pleural cavities are negative.

"*X-ray diagnosis*: Tumor of upper trachea. Chest negative for pulmonary or skeletal metastasis or other pathology."

Patient had with him a tumor mass that he said had been coughed up on July 27, 1943. It was a smooth oval tumor about the size and shape of a large almond and showed a small area where it had formerly been attached. This was submitted to Dr. Violet Keiller who pronounced it a fibrosarcoma.

On account of the dyspnea which had come on so rapidly immediate operation was advised, but this was delayed for two days in order for his father to come to Houston.

Operation: On December 11, 1943, under local anesthesia, the tumor was exposed by direct laryngoscopy and found to be at-

tached to the anterior wall in the region of the cricoid isthmus. The greater part was removed with a snare and the base smoothed with punch forceps. The attachment was anterior and to the right and seemed to include the cricoid isthmus and the tracheal wall for a short distance. There was comparatively little bleeding.

The patient suffered very little reaction and relief of dyspnea was complete. Three days later the application of a radium pack to the neck over the site of the tumor was begun. Fifty mg. of radium were applied for 14 hours on three successive nights. My reason for the use of the radium was that, although I had never before seen a fibrosarcoma in this region, I had seen three in other locations that recurred promptly after thorough removal, but which remained well when removal was followed by the radium pack. One of these grew from the periosteum of the orbit and two from the periosteum of the bones of the face.

Healing in this case was uneventful except that the site of attachment showed granulations for some time. When healing was complete there remained a small, smooth, slight elevation which is still present. He has reported regularly for observation and there has been no sign of recurrence.

The following are the pathological reports:

1. (From the Laboratory of Torney General Hospital) *Gross Description*: The specimen consists of a piece of tissue measuring 2 x 1 x 0.7 cms. Part of its surface appears to be covered by mucosa which is smooth. The consistency is moderately firm. On cross section, it is composed of grayish-white tissue which is homogeneous in structure.

Microscopic Description: The sections show the mass to be composed of interwoven strands of spindle cells and fibers which are densely packed. The fibers are not well defined, they appear rather coarse and sometimes show suggestions of longitudinal striations. Often the fibers seem to form a syncytium. The ends of the fibers are not clearly visualized but they seem to be pointed. The nuclei are large, oval in shape, and vesicular in type. The nucleoli are plainly visible and are frequently eosinophilic. The nuclei vary in size, shape and staining qualities but mitotic figures are rarely encountered. Amitotic division is often suggested and quite a few composite giant nuclei are seen. The vessels are moderately numerous and are of the capillary type. There is no necrosis. A few groups of lymphocytes are scattered about and polymorphonuclear leukocytes are not uncommon. The surface is covered with nonkeratinizing stratified

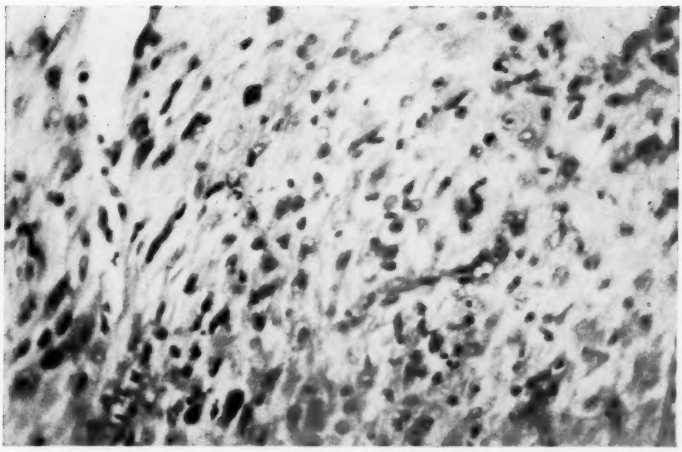


Fig. 2—Photomicrograph, Fibrosarcoma of the Larynx—High Power Section.

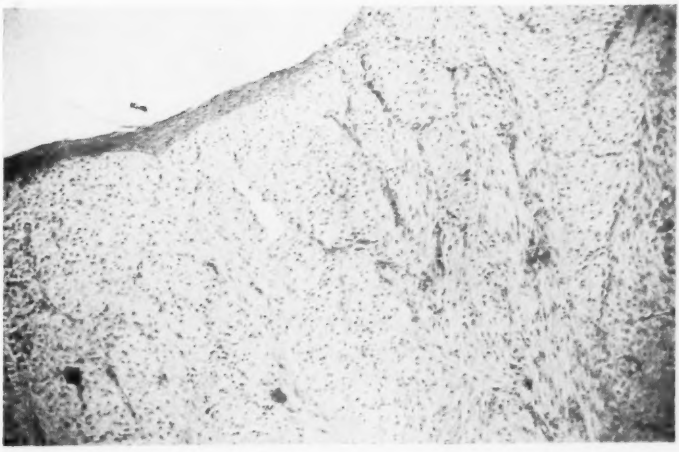


Fig. 3.—Photomicrograph, Fibrosarcoma of the Larynx—Low Power Section.

squamous epithelium which is stretched and thinned out; it appears intact except for a few places where it is replaced by fibrinopurulent exudate and granulation tissue.

P.T.A. Stain: This adds nothing of note. No cross striations of the fibers are seen.

Note: The absence of clinical symptoms in the presence of a mass of this size in the larynx seems very remarkable. Histologically the picture is that of a tumor of connective tissue origin which is quite cellular and actively growing. There is lack of invasiveness—as seen by the intact surface epithelium—but the irregularity of the nuclei, the mitotic figures and suggestions of amitotic division warrant close observation of the patient regarding recurrence of this growth.

Diagnosis: Cellular fibroma of larynx. Fibrosarcoma?

2. (From Col. J. E. Ash, American Registry of Pathology, Army Medical Museum). This is histologically a fibrosarcoma but it has been our experience that these tumors are not so prone to metastasize. Complete surgical eradication is the only recommendation that we can make as regards treatment.

3. (From Dr. Violet Keiller, Houston). *Gross Description*: The material consists of two specimens; the first of these is a tumor mass, brought by the patient, December 8, 1943, with the history that it had been coughed up in July, 1943. The second specimen is tumor tissue removed from the subglottic region by Dr. Foster, December 11, 1943.

The first specimen has been preserved in formalin and is in perfect condition. The tumor is a long ovoid, 2.3 cm. in length, with a curious knob turned up at one end, 1.5 cm. in height; this seems to have been the pedicle of attachment of the tumor. The surface is smooth, and the interior firm, rather soft, homogeneous, white from long fixation. Immediate section showed it to be a fibroblastic sarcoma.

The second specimen is in several pieces, the largest of them 1 cm. in diameter. The entire mass is estimated as 2 cm. by 1.5 cm. by 1 cm. or slightly larger. The tissue is greyish-pink, homogeneous, soft but not friable; the appearance of the fresh cut section suggests sarcoma.

Microscopic Description: Section through the length of the first specimen, excluding the "pedicle", shows the tumor to be cov-

ered by thin stratified squamous epithelium, reduced by pressure in some areas to two or three layers of cells, showing no proliferative changes, and with a clearly defined base. There is no ulceration of the surface, nor is there any inflammatory reaction in the depths of the tumor. This is composed of interlacing bundles of active young fibroblasts, in the form of long spindles, with long oval nuclei, of about the same size. Small vessels with thin but adequate walls are present in moderate number.

In the second specimen the relation of tumor to surface epithelium is similar to that described in the first specimen. Again there is no ulceration of the surface, and while in this case there is slight inflammatory infiltration throughout the entire mass, there is no tendency for the infiltrating cells to concentrate near the tumor surface.

The pattern of the tumor tissue has changed but little since the formation of the first tumor; the most noticeable alteration is in the blood supply. Many more small blood vessels are present and some of these are channels with imperfect walls. No alteration is observed in the rate of growth of the essential tumor cells, nor in the anaplastic character of these, although some increase in the degree of malignancy would have been expected as this is a recurrence, following operative removal after the spontaneous extrusion of the first tumor.

Diagnosis: Fibroblastic sarcoma of larynx, grade 2.

COMMENT

This case is reported for two reasons:

1. A fibrosarcoma in this region is sufficiently rare to warrant reporting.

2. The use of radium following removal of fibrosarcomata has been so satisfactory that I feel that it, too, should be recorded.

1304 WALKER AVENUE.

LXXV

ARNOLD'S NERVE REFLEX COUGH SYNDROME

HAROLD I. LILLIE, M.D.

AND

WILLIAM C. THORNELL, M.D.

ROCHESTER, MINN.

All gradations of nonproductive cough may be observed, from an occasional cough, accompanied or preceded by a sensation of tickling or a sensation that a foreign substance is in the pharynx, on to the more severe paroxysms of coughing which may be followed by syncope and are variously designated as "laryngeal vertigo", "laryngeal epilepsy" or "laryngeal syncope."

In routine examinations, it frequently is noted that certain normal individuals are caused to cough when the examiner touches that portion of the external auditory canal which is supplied by Arnold's nerve, the auricular branch of the vagus. It has also been noted that loose scales, cerumen, or foreign bodies in the external auditory canals of patients with extremely hyperactive Arnold's nerve reflexes may produce the symptoms enumerated by means of reflex irritation transmitted over the laryngeal nerves.

In 1876 Charcot[1] first introduced the term "laryngeal vertigo" for the symptom complex in which unconsciousness or giddiness was preceded by a paroxysmal cough initiated by a tickling sensation in the throat. He attributed this condition to reflex irritation of the laryngeal nerves and believed it to be similar to the vertigo present in Ménière's symptom complex. Several excellent reviews are to be found in the literature, those of Wilson,[2] Adams,[3] and Whitty[4] are especially illuminating.

A number of theories have been advanced as to etiology. The following classification of Whitty presents the various etiologic factors which have been suggested in the past:

Paper presented at the Minnesota State Medical Society Meeting, Rochester, Minnesota, April 13-15, 1944.

Neurologic causes.

1. Reflex irritation.
2. Epileptic episode.

Cardiovascular causes.

1. Cerebral anoxemia.
2. Venous congestion.
3. Cardiac asystole.

In 1936, Adams[5] reviewed four cases in which this symptom complex was present, and, in one of the cases, he thought that inhalation of dust or tobacco smoke was the exciting cause. Molloy,[6] in 1939, presented a case in which there were symptoms typical of laryngeal vertigo; aneurysm of the aorta, with resulting compression of the esophagus and trachea, was present. It is interesting to note the great frequency with which this condition is found among obese men of middle age who demonstrate a tendency toward elevated blood pressure.

We recently have observed a patient with symptoms of paroxysmal cough, preceded by a sensation of tickling in the throat and followed by syncope, which we believe can be classified under Arnold's nerve reflex cough syndrome. To our knowledge there is no report in the literature referring to a hyperactive Arnold's nerve reflex as being an etiologic factor in so-called laryngeal syncope.

REPORT OF A CASE

A man, 41 years of age, was first seen at the clinic January 24, 1944, complaining of recurrent attacks of coughing and hoarseness. The coughing had developed three years before, following bilateral acute suppurative otitis media. In May, 1941, a tonsillectomy had been performed without any effect on the paroxysms of coughing. In October, 1943, following several severe episodes of coughing, hoarseness had developed and persisted. A sensation of a "plug in the throat" had been noted and coughing failed to relieve the sensation. Various means of medication had been tried but had had no effect on the symptoms. In November, 1943, during one period of eight hours the patient had had 24 paroxysms of coughing, followed by syncope lasting several seconds and occasionally clonic movements of the arms had developed. These symptoms had continued up to the time of his visit to the clinic and had prevented the patient from

continuing with his work. In November, 1943, when he had severe paroxysms of coughing, the patient had been quarantined because he had been thought to have whooping cough.

General physical examination at the time of the patient's admission to the clinic revealed mild hypertension and obesity. Neurologic examination and examination of the nose and throat gave negative results. On examination of the ears, several small scales and a slight amount of cerumen were found. It was noted that stimulation of the area in the external auditory canals supplied by Arnold's nerve reproduced the symptoms enumerated.

Silver nitrate (50 per cent and 20 per cent) was applied several times to the area in the external auditory canal innervated by Arnold's nerve until stimulation of the area failed to produce the symptoms. On February 5, 1944, the patient was dismissed from our observations markedly improved.

The man remained completely free of symptoms for two weeks and returned to the clinic on March 28, 1944, for further treatment because of the recurrence of the paroxysm of coughing. Syncope had not followed the coughing spells after the treatment employed on his first visit.

Examination at this time revealed trigger areas situated in the right external auditory canal superiorly, and in the left canal inferiorly. Much improvement in symptoms was again noted following application of silver nitrate to these areas. The patient was still under observation at the time this report was written. If the improvement in symptoms does not last long, use of electrocautery or injection with alcohol of Arnold's nerve will be considered.

COMMENT

The trigger sensory area involved in the production of Arnold's nerve reflex cough symptom complex is the posterior and inferior portion of the external auditory canal and the posterior half of the tympanic membrane. This area receives its sensory nerve supply from Arnold's nerve, the auricular branch of the vagus nerve. This nerve takes its origin from the superior, or jugular, ganglion of the vagus nerve and, after receiving a small filament from the petrous ganglion of the glossopharyngeal nerve, passes behind the jugular vein and enters a small canal on the lateral wall of the jugular fossa. It then passes through the temporal bone, crossing the facial nerve on its medial surface, and reaches the auricular fissure between the mastoid process and the external auditory meatus. It then divides into two

branches, one communicating with the posterior auricular nerve, while the other is distributed to the external auditory canal and tympanic membrane, as has been described, and to the skin on the posterior surface of the auricle. In some cases it may enter the facial canal and joining with the facial nerve, may then reach its destination over the posterior auricular nerve.

All gradations in this syndrome may be encountered. The case presented represents the extreme manifestation and fits into the large group of cases designated as "laryngeal vertigo." No attempt is made to explain the underlying pathologic changes which may produce these symptoms. In our case what effect the antecedent bilateral suppurative otitis media had on the skin of the auditory canals is not readily explained. There was no physical evidence of pathologic changes.

It would be well to keep in mind this syndrome in all cases of chronic cough in which general physical examination gives negative results. Elicitation of the symptoms by stimulating the trigger area should establish the diagnosis.

MAYO CLINIC.

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The Scientific Papers of the American Broncho-Esophagological Association

PRESENTATION OF SPECIMENS AND NEW INSTRUMENTS

LXXVI

VITALIUM LARYNGEAL MOLD

LEIGHTON F. JOHNSON, M.D.

BOSTON, MASS.

In two cases of laryngeal stenosis, this instrument has proven of definite usefulness to us.

It presents two qualifications to recommend it:

First, it is tolerated by the laryngeal tissues, without demonstrable reaction for indefinite periods. We have left it lying in for periods up to three months.

Second, it has an airway which has proven to be a psychological asset to our patients and could well prove to be a life-saving factor in the event that the tracheotomy tube ceased to function.

LXXVII

A NEW MAGNET FOR THE REMOVAL OF FOREIGN BODIES FROM THE FOOD AND AIR PASSAGES

MURDOCK EQUEN, M.D.

ATLANTA, GEORGIA

I wish to present a magnet, perfected by the General Electric Company, which I believe will prove of great value in the removal of foreign bodies from the tracheobronchial tree, the esophagus and particularly the stomach, since foreign bodies of this type have not been very amiable to conventional esophagoscopy.

This magnet is an alloy of aluminum, nickel, cobalt and iron, and derives its name from the abbreviations of these metals, alnico. It is used as a stabilizer in airplanes and is highly magnetic, being able to lift 25 times its normal weight.

Two types of magnets are presented, one to be used in the removal of magnetizable objects from the tracheobronchial tree and the other to be used in the stomach. The first magnet, (Fig. 1), is attached to an urethral catheter and may be passed through the bronchoscope, under fluoroscopic guidance, and the object removed. The other magnet, (Fig. 2), is attached to a small Levine tube through which a metal stylet has been passed to strengthen the tube. The other end of the Levine tube is attached to a rubber bulb, diaphragm and catheter, this appartus being used to inflate the stomach. The magnet is passed through the mouth and the esophagus into the stomach, under fluoroscopic guidance, and the stomach is inflated. This inflation lifts away any collapsed portion of the stomach from the object and permits the magnet to be passed around in the stomach with ease until the object comes into the magnetic field and is attracted by the magnet and removed.

Only a minority of foreign bodies of the food and air passages are magnetizable, but for those which are, the use of this instrument will render unnecessary many abdominal operations and bronchial instrumentations.

PONCE DE LEON EYE AND EAR INFIRMARY.

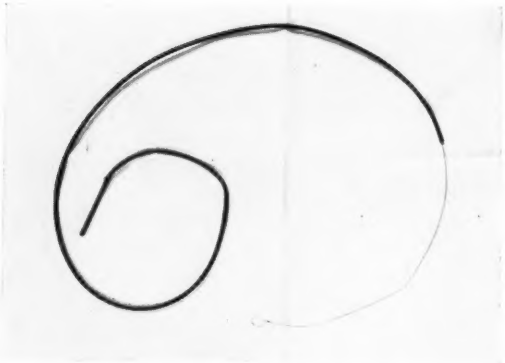

Fig. 1.—The magnet, of cast alnico, is 3.5 cm. in length and 0.5 cm. in diameter, and is attached to a urethral catheter for insertion in the trachea or the bronchus.

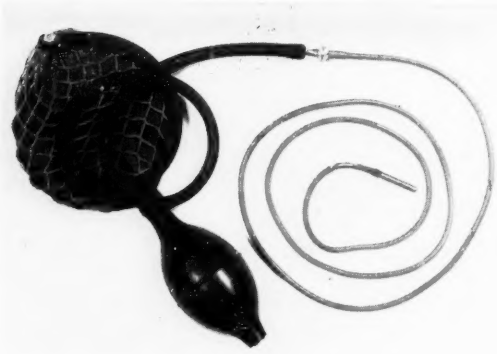

Fig. 2.—The stomach is inflated with air by compression of the rubber bulb attached to the diaphragm and catheter. This inflation lifts away any collapsed portion of the stomach from the foreign body, allowing the magnet to be passed about freely, and the object is unimpeded in being attracted to the magnet.

LXXVIII

RECENT TRENDS IN THE BRONCHOLOGIC USE OF CHEMOTHERAPEUTIC AND BIOTHERAPEUTIC AGENTS

GABRIEL TUCKER, M. D.

JOSEPH P. ATKINS, M.D. (by invitation)

PHILADELPHIA, PA.

In the treatment of diseases which are of primary interest to the broncho-esophagologist, there are several time-honored and fundamental principles which are controlling factors so far as bronchologic therapy is concerned. Among these one might mention the importance of maintaining an adequate cough reflex, the importance of an adequate airway and the importance of the mechanical relief of obstruction caused by abnormal tissue or abnormal secretion. In the therapy of most diseases of the respiratory tract, the utilization of general medical measures in the treatment of infection cannot be neglected. Much change has occurred in recent years with the advent of new chemotherapeutic agents in the medical care of many infections. It is not our purpose in this paper to consider the problems of chemotherapy in general or to consider the medical aspects of the treatment of these diseases. We would prefer to point out the present attitude concerning the use of these several agents only in so far as they particularly affect the structures in which this Society is primarily interested. The use of these agents in no way negates or supplants those fundamental bases on which bronchologic therapy rests. Chemotherapy and biotherapy are adjuncts to the treatment of bronchopulmonary disease rather than panaceas.

Of the biotherapeutic agents we wish to mention but two, both of which are old friends. The first of these is autogenous vaccine used in connection with chronic infections of the tracheobronchial tree. We have found that the use of a carefully prepared autogenous vaccine made from the uncontaminated organisms bronchoscopically collected from the tracheobronchial tree materially enhances the effect of treatment in these cases. Indeed, a great many patients with chronic tracheobronchitis will make an excellent recovery when adequate mechanical drainage of the tracheobronchial tree is achieved and the patient's resistance is stimulated by the autogenous vaccine.

The mechanism of this response is not clearly understood and has been much debated. We do not propose to enter into the debate but simply to attest to the usefulness of the method in our experience.

Even more striking is the response in those allergic patients in whom infection of the tracheobronchial tree acts as an etiologic factor in the production of their symptoms. In these patients the use of bronchoscopically collected material for autogenous vaccine together with bronchoscopic aspiration has been responsible for some of our most gratifying therapeutic achievements. It must be mentioned in this connection that the entire allergic picture must be considered. Relief cannot be obtained from removing a single agent from a patient who has multiple sensitivity.

The other biotherapeutic agent to which we wish to call attention is epinephrine in conjunction with bronchoscopy in the asthmatic patient. For a great many years bronchoscopic aspiration has been used in our clinic for the mechanical relief of bronchial obstruction in severe asthmatics. The obstruction is caused by the accumulation of viscid secretion which they are unable to expel, plus the characteristic spasm of the bronchi. After several experiments in which the tracheobronchial tree of asthmatic patients was aspirated as thoroughly as possible, the patient was then given an appropriate dose of epinephrine. When the bronchial spasm was relaxed by the action of the drug, a further quantity of secretion was released. The relief obtained was considerably enhanced. It is now our routine procedure to give asthmatic patients a dose of epinephrine about ten minutes before the bronchoscopy. The dose used should be slightly larger than that usually given for the relief of the patient's symptoms. This has caused no unfavorable reactions and has made thorough aspiration much more certain. The improved pulmonary drainage tends to relieve bronchial infection where this factor exists.

Sulfonamides.—In 1941, Tucker[6] reported our experience with sulfonamides used locally in the tracheobronchial tree. We have continued to use these drugs since that time. At present we prefer macrocrystalline sulfanilamide and microcrystalline sulfathiazole. The powder is blown into the lung through the bronchoscope using the Clerf powder insufflator. The adult dose seldom exceeds two grams.

Our own experiments on dogs, with both chronic and acute infections, conform with those of Chapple and Lynch[1] on mice in the conclusion that these drugs can be introduced into the lung without injury to the lung and are absorbed into the blood stream from the

lung. The drug disappears from the blood stream in 24 to 48 hours following a single intrabronchial insufflation in either dogs or man.

Examination of the bronchial secretion in patients taking sulfonamides by mouth discloses a sulfonamide level which is approximately one-half that of the blood level. Observations have been carried out with Dr. Harrison Flippin in connection with his studies on sulfamerazine and sulfadiazine and this relationship has been found to be fairly constant. Lell has made observations on sulfanilamide, sulfapyridine and sulfadiazine with similar results.

In the selection of patients on whom sulfonamide medication was to be used locally, it was felt that it was improper and unwise to deny them the benefit of systemic sulfonamide medication in order to observe the local action. Therefore, patients were chosen with low-grade or chronic infections of the tracheobronchial tree which were proving resistant to the usual measures for the treatment of such cases. This included patients requiring surgery for the relief of bronchiectasis where an active infection of the involved lung made control of the local process desirable. In general, it may be said that it has been our experience that a sulfonamide applied locally in these cases tends to reduce the intensity of the inflammatory reaction and inhibits the formation of granulation tissue in the bronchus. The reduction of edema and obstructing granulations improves bronchopulmonary drainage.

It is, of course, true that patients with chronic infections when closely supervised will tend to improve, but we feel that after discounting this fact the improvement which has been observed in the patients treated must be attributed at least in part to the local action of the sulfonamides. We have seen one woman with a bronchiectasis entirely too extensive for any surgical procedure whatsoever. She was complaining of constant hemoptysis which on one occasion was so severe as to require pneumothorax for its control. Remarkable improvement followed the use of sulfanilamide insufflation. Granulations which had previously filled the tracheobronchial tree disappeared and the bleeding stopped. Her bronchiectasis was not cured, but she was able to return to useful work and has been getting along very satisfactorily.

A young man of 22 years of age had bronchiectasis involving the entire left lung. Satisfactory control of the bronchial infection could not be achieved by repeated bronchoscopic aspiration and general medical care. With the addition of sulfanilamide insufflation into the affected lung the inflammatory process improved to the

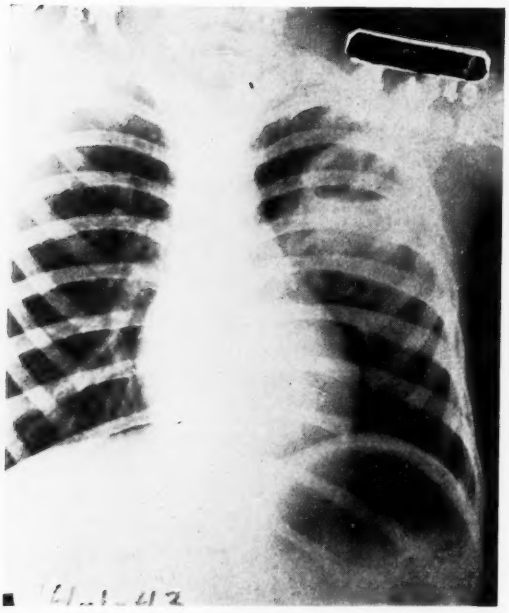

Fig. 1. Case 1.—Roentgenogram of a child four years old in which adequate pulmonary drainage was obtained by bronchoscopic aspiration of the left upper lobe abscess. Pure culture staphylococcus. The infection was unaffected by full dosage of sulfadiazine.

point where surgical intervention was feasible and a very excellent recovery from his pneumonectomy ensued.

The improvement which has been observed is never dramatic and this treatment cannot be used to the exclusion of other medical measures, but it may be a useful adjunct to them. A word must be said concerning the use of sulfonamides as far as their contraindications and possible dangers exist. We have observed one or two patients in whom bleeding from the tracheobronchial tree seemed to follow the use of local sulfonamide medication. The explanation for this is not clear, but we believe that it is due to bleeding from granulations in the bronchial wall. It has never been serious or alarming and has stopped when sulfonamides were discontinued.

We believe that the use of sulfonamides is contraindicated in asthmatic patients with acute symptoms. On one occasion the intro-

Fig. 2. Case 1.—Lateral view of the foregoing.

duction of sulfonamide powder into a patient suffering a mild asthmatic attack produced serious respiratory embarrassment and required a second bronchoscopy to remove as much of the insufflated powder as possible before the symptoms were relieved. This patient had a tracheobronchial infection which was apparently one of the factors producing her symptoms. Subsequent to the insufflation and the subsidence of the acute episode, the patient showed considerable clinical improvement both in regard to her asthma and the local appearances in the tracheobronchial tree.

Inasmuch as we know that sulfonamides used locally in the lung are absorbed into the general circulation both in man and in animals, the use of the drugs carries the danger of sensitization. We have never seen any evidence of sensitivity or toxic reaction resulting from their intrabronchial use but, inasmuch as sensitization has been reported from dermatologic preparations of sulfonamides, there is no reason to think that it could not occur. This may constitute a deterring

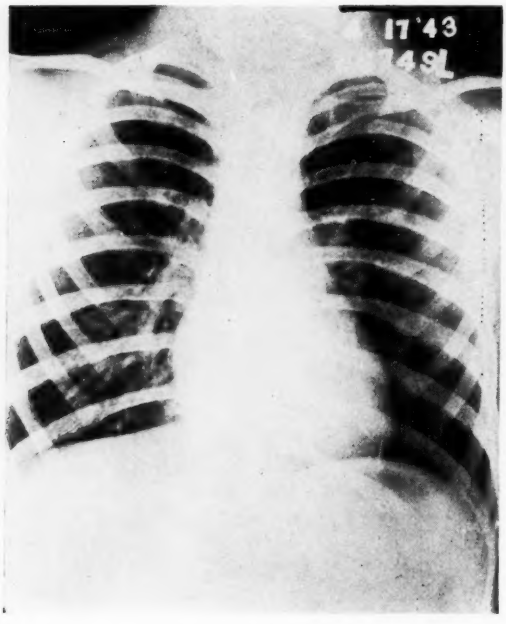

Fig. 3. Case 1.—Roentgenogram of the same child showing complete clearing of lung after 16 days of intravenous penicillin therapy.

element to the local use of this type of medication. Were sensitization to occur, it might become necessary to deny the patient the benefit of sulfonamide therapy in a serious subsequent illness. A number of our patients have received large doses of sulfonamides by mouth following intrabronchial use without signs of sensitivity.

Penicillin.—With the cooperation of Dr. William White of the Harrison Department of Research Surgery of the University of Pennsylvania we have had the opportunity of studying the effect of penicillin in bronchopulmonary infections. The drug has been administered both locally and systemically.

Penicillin has been used locally in the tracheobronchial tree in an aqueous solution containing 500 units per cc. This dosage has been used because stronger solutions have a tendency to produce mucosal irritation.

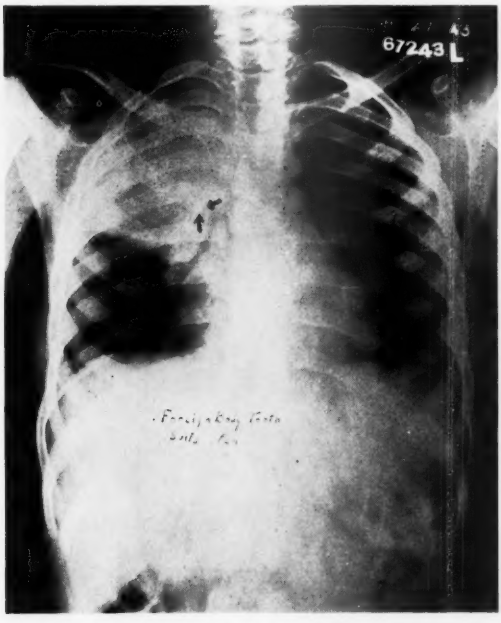

Fig. 4. Case 2.—Roentgenogram showing obstruction of the right upper lobe bronchus by foreign body (tooth) with consolidation of right upper lobe. Unaffected by large doses of sulfonamides and penicillin.

In acute infections of the tracheobronchial tree it has been used in tracheotomized children. In these cases the drug is administered through the tracheotomy cannula in doses of 250 to 500 units every 30 or 60 minutes. The chronic infections treated have been bronchiectasis and purulent tracheobronchitis of older children and young adults. The drug is administered through the bronchoscope at the time of bronchoscopy, or introduced through the larynx in doses of 2500 units.

Our studies indicate that the drug disappears entirely from the tracheobronchial secretions in approximately 24 hours. The effect of penicillin administered locally in the acute infections of tracheotomized children has been to reduce the mucosal swelling and permit more thorough aspiration of the viscid secretion which these patients invariably develop. The secretions appear to be less viscid and more easily removed than those in which sterile water or sodium chloride

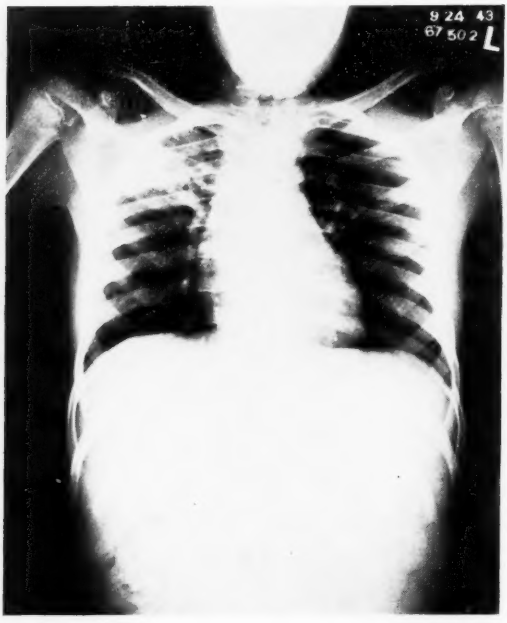

Fig. 5. Case 2.—Roentgenogram showing clearing of right upper lobe following removal of tooth. A similar result would be expected following re-establishment of bronchial drainage if chemotherapy had never been used.

solution was used as a liquifying agent. As a result of the improved drainage, atelectasis was seen less often and was more easily relieved in those patients in whom it occurred. Most of these patients were given penicillin intramuscularly in conjunction with the local administration. The local use of penicillin in no way diminishes the need for aspiration or proper humidification, but it aids in achieving the result these are designed to accomplish. Bronchoscopic aspiration has not been required in any of our treated patients.

We have seen a two-month-old baby admitted to the hospital with left upper lobe consolidation and obstructive laryngeal dyspnea which failed to respond to sulfadiazine. A tracheotomy was done and penicillin given intramuscularly and intratracheally. The child had a profuse flow of bronchial mucus which required frequent aspiration and high humidity. We were able to maintain adequate ventilation in this child throughout the acute episode. Despite the con-

tinued presence of staphylococcus in the bronchial secretion, the organism was sufficiently inhibited to permit the child to make a very satisfactory recovery.

The local use of penicillin in chronic infections of the tracheobronchial tree is not sufficiently advanced to make any certain claims for or against its use. Thus far the effect seems to be similar to that observed with sulfonamides A soluble nontoxic agent having marked antibacterial power offers obvious advantages over the sulfonamides. We have used the drug without observing any unpleasant reactions and believe further study is desirable.

With increasing availability, the number of patients treated systemically with penicillin is rapidly increasing. It has proven itself effective in the control of acute pulmonary infections.

We wish to describe briefly two cases which seem to indicate the place this drug can occupy in relation to diseases requiring bronchologic therapy.

A four-year-old boy developed an acute staphylococcal lung abscess of the left upper lobe. With adequate dosage of sulfadiazine he continued to show systemic signs of marked toxicity, and no improvement in the lung was observed. Bronchoscopic examination disclosed a small amount of granulation tissue in the left upper lobe bronchus. The drainage of the abscess through the bronchus seemed to be adequate. When penicillin was given intravenously, the child promptly improved and proceeded to make an uneventful recovery. (Figs. 1-3).

The second case was an eleven-year-old boy admitted with right upper lobe consolidation and marked systemic signs of infection. He was treated with sulfadiazine and penicillin without affecting his illness. Bronchoscopic examination disclosed almost complete obstruction of the right upper lobe bronchus by mucosal swelling. A large amount of pus was aspirated from this area with a curved aspirator. Following the first bronchoscopy there was little clinical change. The night before a subsequent bronchoscopy, he coughed out a tooth from the lung. The temperature promptly subsided. On re-examination the lumen of the bronchus was again open and the lobe was draining freely. His recovery was complete. (Figs. 4 and 5).

Penicillin can be used to control bronchopulmonary infections. If control of infection alone can restore the normal pulmonary function, it can accomplish results such as were seen in the first case;

otherwise, additional measures will be required for the restoration of a physiologically functioning respiratory tract.

CONCLUSIONS

Autogenous vaccines are useful adjuncts in the treatment of chronic tracheobronchitis and allergic tracheobronchitis.

Epinephrine is a valuable aid in the bronchoscopic aspiration of secretion from asthmatic patients.

Of many drugs which have been used locally in the tracheobronchial tree for the treatment of infection, sulfonamides and penicillin are the first which are both noninjurious to the lung and possess therapeutic effectiveness.

250 S. EIGHTEENTH ST.

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LXXIX

TREATMENT OF CHRONIC NONTUBERCULOUS PULMONARY INFECTION BY BRONCHOSCOPY AND INSUFFLATION OF SULFONAMIDE COMPOUNDS

PORTER P. VINSON, M.D.

RICHMOND, VA.

An increasing number of patients with chronic nontuberculous pulmonary infection are being relieved of symptoms by surgical removal of one or more lobes of the lung. However, the majority of patients with this type of infection either are not suitable for surgical treatment or do not desire operation. Many types of medical treatment have been instituted for these patients, with varying degrees of benefit. Postural drainage, inhalation of various drugs and oral and intravenous administration of medicinal substances, as well as introduction of medicaments into the tracheobronchial tree, have all been employed in the treatment of patients with suppurative pulmonary disease. In recent years secretions aspirated at bronchoscopic examination have been cultured; from these cultures vaccines have been prepared, but their use has been followed by questionable benefit. The tracheobronchial tree has, at the time of bronchoscopic examination, been treated by irrigations or injections of various medicinal agents.

Prior to September 1938 it was my practice to recommend a diagnostic bronchoscopic examination for all patients with chronic pulmonary lesions not tuberculous in origin. However, the results of local therapy of the tracheobronchial tree, other than dilation of bronchial strictures and removal of hemorrhagic granulation tissue, proved quite ineffective in my patients. For this reason I ceased recommending bronchoscopic treatments for patients with pulmonary suppuration without bronchial stricture, although for many of them we employed postural drainage with fairly satisfactory results.

Since September 1938 I have treated approximately 100 patients with chronic suppurative pulmonary disease other than abscess by insufflating, first, powdered sulfanilamide and, later, a mixture of two parts of sulfanilamide and one part of sulfathiazole into the

From the Medical College of Virginia.

tracheobronchial tree, after evacuating as much secretion as possible by suction. The powdered drugs are sterilized as if they were to be used intraperitoneally, and care is taken that the tubes employed for insufflation are thoroughly dried. Even a minute amount of moisture will clog the tube and prevent free passage of the powder into the bronchi. The amount of the substance that is used is apparently unimportant. I insufflate a sufficient quantity of powder to cover the bronchi thoroughly, 3 or 4 gm. usually being adequate.

Evaluation of treatment of any group of patients in whom curative results are not obtained and only palliation is expected will depend, in part, on the enthusiasm of the physician and the hope of the patient. In spite of the uncertainty of accuracy in the report of results in a group of patients of this type, I believe that one-half the number of patients thus treated have been benefited. In a few, results have been spectacular, with great reduction in cough and expectoration, gain in weight and pronounced improvement in the patient's sense of well-being. In the majority of patients, sputum that was previously foul smelling has become less fetid, and almost all patients have shown a reduction in the incidence of bleeding. In patients having asthmatic bronchitis with associated purulent secretion the results have been particularly impressive. Several who were ill from absorption of the products of suppuration have been relieved entirely of cough and expectoration by one or two applications of the powdered sulfonamide compounds into the tracheobronchial tree.

REPORT OF A CASE

A woman, aged 50, was examined August 5, 1943. She was well until two years before, when an acute infection of the respiratory tract developed, followed by attacks of dyspnea at night and after exertion. She gradually became worse and stated that she seemed to "fill up" with secretion, which had become purulent. At the time of her examination here she expectorated a pint (500 cc.) of purulent secretion a day, and she had a daily elevation of temperature to 101° F. There had been a reduction in weight from 180 to 130 pounds (81.6 to 59 Kg.).

Examination of the thorax revealed wheezes over the base of both lungs, with diminished breath sounds. Roentgenoscopic study did not show any significant change. Bronchoscopic examinations on August 5 disclosed a large amount of thick, purulent secretion in the lower lobe of both lungs and intensely inflamed bronchial walls. The pus was thoroughly aspirated, and 3 or 4 gm. of a mixture of

sulfanilamide and sulfathiazole powder was insufflated into the bronchi of both lungs. The patient began to improve immediately, and the temperature became normal within three days after treatment.

On August 12 a second bronchoscopic examination revealed practically no pus, although the bronchial wall showed evidence of slight congestion. She was dismissed from my care a week later; a report from her two months after dismissal stated that she was free from pulmonary symptoms and that she had gained 15 pounds (6.8 Kg.) in weight.

In patients with bronchiectasis who show improvement, the cough and sputum are usually reduced to a third or a fourth of the original amount.

Untoward results have not been noted in any of my patients, and I do not hesitate to employ this method of treatment for any patient with chronic suppurative pulmonary disease. My youngest patient thus treated was 22 months of age. Possibly treatment with other drugs of the sulfonamide group in various combinations might prove beneficial in patients who have not shown improvement after insufflation of sulfanilamide and sulfathiazole. When penicillin becomes readily available, so that it can be utilized in the treatment of chronic infections, local application may provide relief in patients in whom favorable results have not been obtained by the use of sulfonamide compounds. So many varieties of bacteria have been associated with chronic pulmonary infection that specific therapy has not been applicable. Improvement in bacteriologic study of secretion from the tracheobronchial tree may provide information that will increase the efficacy of local therapy.

SUMMARY

Approximately 100 patients with chronic nontuberculous suppurative disease of the lungs, not including pulmonary abscess, have been treated during the past five years by bronchoscopic removal of secretion from the tracheobronchial tree, followed by intrabronchial insufflation of powdered sulfonamide preparations. At least half of the patients thus treated have been benefited, improvement varying from almost complete relief of symptoms in patients with chronic asthmatic bronchitis associated with purulent secretion to about 75 per cent reduction in cough and expectoration in patients with well-established bronchiectasis. No untoward results have been observed

in any of these patients. Refinement in bacteriologic study of secretions from the tracheobronchial tree may provide information as to the type of drug that will prove most beneficial. Further advances in the preparation of chemotherapeutic agents and of penicillin-like substances may add to the efficacy of local therapy for patients with suppurative pulmonary disease.

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LXXX

MEDIASTINAL COMPLICATIONS ASSOCIATED WITH ESOPHAGOSCOPY

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Carcinoma of the Esophagus: Esophagoscopy Followed by Mediastinal Abscess.—Irvin P., colored male 50 years of age, was seen with a history of precordial pain and difficulty in swallowing for the past eight months.

An esophagoscopy was done under local anesthesia. The day after the esophagoscopy the patient's temperature rose to 101° F., and he complained rather bitterly at this time of substernal pain. The next day the patient was unable to swallow fluids; his neck was extremely tender and swollen on the right. Examination of the chest was entirely negative. His temperature ranged from 100° to 103° F.

The neck became more swollen and tender, but at no time was there any emphysema present to palpation. A chest plate revealed an area of increased density extending from the right hilum. A lateral film of the neck revealed an extensive accumulation of air just anterior to the cervical vertebra. On the fifth day the patient suddenly became very dyspneic and expired before a tracheotomy could be done.

Autopsy report: Abscess of the right mediastinum and the walls of the esophagus. Carcinoma of the esophagus.

Esophagoscopy for Removal of Chicken Bone Complicated by Mediastinitis. External Drainage in Neck. Cure.—Mrs. C. A. McS., 59 years of age, was seen with a history of having swallowed a chicken bone 24 hours previously. She was unable to eat or drink.

An esophagoscopy was attempted under local anesthesia but the patient complained of pain and was not cooperative; therefore, ether was given and the esophagoscopy done. No foreign body was seen. At the end of the esophagoscopy a note was made of bleeding from this region. Within an hour after completion of the operation, the patient complained of pain in her back and deep in her chest, especially on the left side. X-ray examination showed considerable sub-

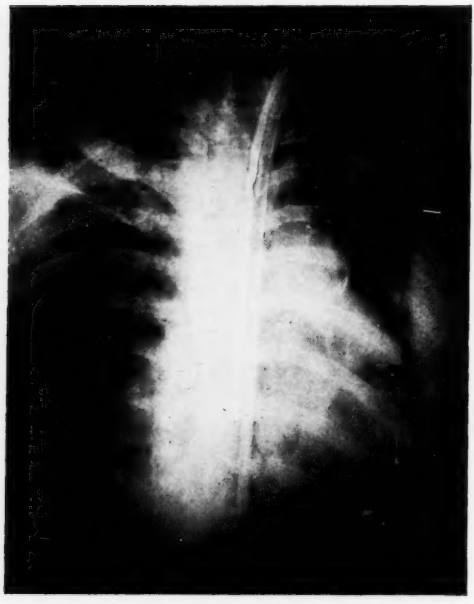

Fig. 1. Case 2.—Roentgenogram showing chest pathology and drainage tubes.

cutaneous air in both sides of the neck and also in the mediastinum where it was particularly well shown around the aortic arch. There was some elevation of the right diaphragm with atelectasis of the right lower lobe.

Eight hours after the operation an incision was made anterior to the left sternomastoid muscle and by blunt dissection the left side of the esophagus was exposed. About two inches below the cricoid in this region, one ounce of straw-colored fluid was encountered. There was a large opening into the posterior mediastinum from which more fluid was draining. No foreign body or perforation of the esophagus was seen. Two rubber tubes were placed deep in the posterior mediastinum with the application of sulfathiazole powder and the wound was packed with iodoform gauze. Sulfadiazine and glucose solution were given intravenously. Nothing was given by mouth for ten days. The highest temperature was 102° F.

The patient made a rather slow but satisfactory recovery.

899 Madison Avenue.

LXXXI

PERFORATION OF LOWER END OF THE ESOPHAGUS; CHEMOTHERAPY, GASTROSTOMY; RECOVERY

CLYDE A. HEATLY, M.D.

ROCHESTER, N. Y.

A perforation in any portion of the esophagus presents immediate problems of unusual gravity which call for prompt yet carefully considered methods of management. This is particularly true in perforations involving the lower third of the esophagus where the spread to surrounding structures is frequently rapid and there is little tendency to localization and where the technical difficulties of successful surgical drainage are correspondingly great. The prompt use of chemotherapy in conjunction with cessation of mouth feedings and subsequent gastrostomy offers a plan of management which may prove helpful in many of these serious emergencies.

REPORT OF A CASE

P. K., a man of 45 years of age, was admitted to the Strong Memorial Hospital on June 21, 1943, with the complaint of dysphagia of 15 months' duration together with a weight loss of 25 pounds. Fluoroscopic and x-ray studies showed an obstruction at the level of the diaphragm which was thought to be a cardiospasm. There were no significant findings in either the general physical or the laboratory studies.

The following day, an esophagoscopy was performed under ether anesthesia. The patient was extremely nervous and apprehensive, and a general anesthesia was considered advisable. The findings were those of cardiospasm. There was no evidence of associated stricture. A Tucker pneumatic dilator was introduced through the esophagoscope, and gentle dilatation carried out to somewhat less than the full capacity of the bag. Inspection of the diaphragmatic area showed no signs of trauma following the withdrawal of the dilator.

Seven hours later, the patient complained of sudden severe pain in the substernal region and shortly after of chilly sensations. A sharp temperature rise to 39.5° C. was reported. No changes could be detected in the lungs on physical examination nor was any abdominal tenderness or rigidity observed. The epigastric pain continued during the night and by morning had spread to the left scap-

ular area. The leucocyte count at this time was 48,000 with 95 per cent polymorphonuclears. An x-ray film showed extensive emphysema extending throughout the mediastinum and into the neck as well as clouding of both lung bases. A few hours later, the emphysema in the subcutaneous tissues of the neck was clinically demonstrable. The fever remained continuously elevated between 39° and 39.5° C. These findings were interpreted as indicating a perforation in the lower third of the esophagus above the level of the diaphragm, with mediastinitis.

In view of the diffuse changes visible in the x-ray film without evidence of localization, it was felt that the patient's best chance for recovery lay in intensive chemotherapy. All mouth feedings had, of course, been discontinued with the onset of pain and fever. Accordingly, two infusions of 1000 cc. of Ringer's solution, each containing 5 gm. of sulfadiazine were given subcutaneously on the first postoperative day. The following day, the patient was given a similar amount subcutaneously and intravenously. The third day, he received two subcutaneous infusions of 1000 cc. each of Ringer's solution with 5 gm. of sulfadiazine in addition to 1000 cc. of 5 per cent glucose intravenously. The blood level of sulfadiazine was identical on the second and third days—21.7 mg. per 100 cc. A white blood cell count taken on the third day was 14,800.

The patient appeared clinically improved, and on the fourth postoperative day, a gastrostomy was performed. Sulfonamide therapy was continued maintaining a blood level between 21 and 22 mg. per 100 cc. An x-ray film taken on the sixth day showed the emphysema largely disappeared with the shadow at the right base somewhat increased in density and with some changes at the left base as well. An x-ray film taken on the tenth postoperative day showed progressive clearing and the roentgenologist observed that it now appeared that the mediastinal and pleural infection might well resolve without the formation of an abscess. The sulfonamide therapy was discontinued on the twelfth postoperative day because of the presence of a few red blood cells in the urine. By this time, the temperature had become normal. On this same day, water by mouth was begun in small amounts. Five days later, a soft diet was started. The gastrostomy tube was removed on the twenty-third day, and the patient discharged from the hospital. He had been fever free for ten days and was swallowing without difficulty. He has continued well during the past year, has regained most of his weight, and reports only occasional dysphagia.

11 North Goodman Street.

LXXXII

IMPACTED FOREIGN BODY IN THE ESOPHAGUS REQUIRING EXTERNAL OPERATION FOR REMOVAL

JOHN H. FOSTER, M.D.

HOUSTON, TEX.

G. M. M., aged five years, was brought to the office on June 30, 1943. The history given was that almost an hour and a half previously, while playing with a marble, the child swallowed it and choked. There was some dyspnea and the mother attempted to remove the marble with her finger, but was unsuccessful.

The child was seen about 5:00 p. m. by Dr. J. H. Barrett. There was slight respiratory difficulty but the child was not cyanotic. Examination of the nose and throat was essentially negative except for a slight amount of hemorrhage due to abrasions of the mucous membrane of the throat. There was no evidence of foreign body in the oropharynx or hypopharynx, but there were contusions in the latter. Examination of the chest was negative except for slightly labored breathing which seemed to be due to pressure on the trachea.

An esophagoscopy was decided upon and the child was admitted to the Houston Eye, Ear and Throat Hospital. Under ether anesthesia an esophagoscope was introduced and a moderately large glass marble was found in the upper part of the esophagus. The foreign body was tightly wedged in the esophagus and it was impossible to get forceps around it. Equally unsuccessful were attempts to push the marble down. At about this time I came into the hospital and Dr. Barrett asked me to see the case. I was unable to dislodge the marble.

We then told the father that we felt an external operation would be necessary. Dr. George Waldron, a general surgeon, was called into consultation and operation agreed upon, but it was decided to postpone this until the next morning.

At 10:00 a. m., July 1, 1943, another attempt was made to remove the marble by esophagoscopy but this was again unsuccessful. The esophagus was then exposed through an incision along the anterior border of the sternomastoid muscle. When the foreign body was located, an attempt was made to push the marble up or down by grasping the esophagus between the fingers but it was impossible to

move it. An incision was made in the esophageal wall and the marble removed. There seemed to be nothing unusual in the structure of the esophagus. The wound in the esophagus was closed and the neck wound sutured with drains. There was considerable postoperative drainage, but otherwise recovery was uneventful.

COMMENT

This case is reported for the reason that I have never seen one like it and I have talked with no one who has. Frankly I did not believe it possible for a round, smooth marble to be so tightly impacted as to defy any attempt to dislodge it even by manual manipulations after exposure of the esophagus.

The most plausible explanation would seem to be that the marble was lodged in a split of the fibers of the cricopharyngeus muscle but we were unable to demonstrate this.

1304 WALKER AVENUE.

LXXXIII

FATAL HEMORRHAGE FROM PERFORATION OF RIGHT RENAL ARTERY BY FISH BONE

HAROLD LESLIE KEARNEY, M.D.

NEW ORLEANS, LA.

Hemorrhage from erosion of large vessels following ingestion of sharp-pointed foreign bodies is fortunately comparatively unusual. Clerf[1] reports a fatal case of hemorrhage from the innominate artery from perforation by a safety pin in a 22-month-old child, and another case of fatal hemorrhage from perforation of the posterior wall of the left ventricle by a safety pin in a two-year-old child; both these cases were esophageal foreign bodies. Tucker[3] reports a fatal hemorrhage from erosion of a safety pin through the esophageal wall into the aorta in a seven-month-old infant. Holinger[2] reports a case of fatal hemorrhage from the aorta in a seven-month-old infant in whose stomach an open safety pin was found at postmortem examination.

Mr. H. K. G., age 44 years, came to my office on August 11, 1943, complaining of intense pain between the scapulae since swallowing a fish bone 24 hours before. A skiagraph of the chest showed no widening of the mediastinum and no radiopaque foreign body of the esophagus. Total white blood cells were 10,300 with 88 per cent neutrophiles. Mirror examination of the hypopharynx and larynx was negative. The patient was edentulous. The symptoms suggested perforation of the esophagus with impending mediastinitis.

A careful search of the entire esophagus with a large lumen esophagoscope showed no evidence of either foreign body or trauma. The following day he had no pain on swallowing and was discharged.

On October 21, 1943, more than two months later, he was readmitted to Touro Infirmary to the service of Dr. Lewis H. Levy with pain in the lower abdomen radiating to the right lumbar region. This illness had begun six days before and was accompanied by several chills and with irregular fever to a maximum of 104° F. Examination showed him to be well developed, well nourished, pale, sweating profusely and evidently acutely ill. Cystoscopic examination by Dr. W. A. Reed showed the bladder and the ureters negative;

Fig. 1.

the pyelo-ureterogram was normal except that fixation of the right kidney was indicated. At laparotomy by Dr. Levy a huge retroperitoneal clot was found; there was a rather definite odor of urine. The source of the bleeding could not be found. The patient died October 25, 1943.

At autopsy an opening 6 x 5 mm. was found in the right renal artery. Protruding from this opening was a bone 3 cm. in length by 2 mm. at its widest portion. (Fig. 1.) No perforation of the esophagus or gastro-intestinal tract was found to indicate the path of the needle-like foreign body to the renal artery. The portion of the alimentary tract nearest the right renal artery is the duodenum and it is probable that the perforation took place here and healed before erosion of the artery occurred.

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LXXXIV

BENIGN TUMOR OF THE ESOPHAGUS

HERMAN J. MOERSCH, M.D.

AND

STUART W. HARRINGTON, M.D.

ROCHESTER, MINN.

Benign tumor of the esophagus is of special interest because of its comparative rarity and the fact that it may attain considerable size without the production of esophageal symptoms. The possibility of benign tumor of the esophagus must be considered in every case of dysphagia and care must be exercised to distinguish benign from malignant tumor of the esophagus, which is relatively common. This is important from a therapeutic standpoint, for benign tumors of the esophagus are extremely amenable to treatment while malignant tumors are very resistant to treatment.

The comparative rarity with which benign tumor of the esophagus occurs is well illustrated by the studies of Patterson.[1] In a review of the literature between 1717 and 1932, she found records of only 62 such cases, including those in which the tumor was first recognized at necropsy. The rarity of benign tumor of the esophagus is further emphasized in the study of Vinson, Moore and Bowing,[2] who, in a review of 4,000 cases in which patients were seen at the Mayo Clinic because of dysphagia, reported that they were able to find only three cases of benign tumor of the esophagus. Since their report, approximately 7,000 additional patients suffering from dysphagia have been seen at the Mayo Clinic and in this group twelve additional cases of benign tumor of the esophagus have been found, bringing the total to 15 out of 11,000 patients who had dysphagia.

If only patients who give a history of dysphagia are studied, one obtains an inaccurate picture of the true incidence of benign esophageal tumor, for it is well known that many benign tumors of the esophagus do not give rise to symptoms. This is well exemplified in a review of 7,459 postmortem examinations performed at the Mayo Clinic, in which 44 benign tumors of the esophagus were found. In none of the cases was there a history of esophageal difficulty. The character of these tumors is listed in Table 1.

TABLE 1.

TYPES OF BENIGN TUMOR OF THE ESOPHAGUS
FOUND IN 7,459 NECROPSIES

Cysts	2
Hemangioma	3
Leiomyoma	32
Mucocele	1
Neurofibroma	1
Papilloma	3
Polyps	2
Total	44

A review of the literature reveals a great variety of benign tumors that may originate in the esophagus. Among these may be mentioned adenoma, aberrant thyroid, cyst, fibroma, hemangioma, leiomyoma, mucocele, myoma, myxofibroma, neurofibroma, papilloma and polyp. Most authors state that polyps are the most common type of benign tumor of the esophagus. Our experience is similar to that of Rose,[3] that leiomyoma is the most common type of benign tumor of the esophagus.

The present study is based upon a review of data on the 15 cases of benign tumor of the esophagus in which the diagnosis was based on clinical findings. The character of the tumors in this group is listed in Table 2. The tumors listed as of indeterminate type were most likely leiomyomas but because of lack of microscopic confirmation they are classified as "indeterminate."

As has been pointed out by other observers, benign tumor of the esophagus is more common among men than among women. Eleven of our patients were men and four were women. It is of interest, however, that when the benign tumor was of intramural, extramucosal type the incidence was divided equally as to sex. Benign tumor of the esophagus is found in all age groups but is encountered most frequently after the fourth decade. Only three of our patients were less than 40 years of age.

For purposes of description benign tumors of the esophagus are divided into two types, depending on their site of origin. Tumors of the first type arise from the mucosa or submucosa of the esophagus

and are commonly known as intra-esophageal or mucosal tumors. Tumors of the second type have their origin in the outer coats of the esophagus and are best described as intramural, extramucosal lesions. In our group, nine of the tumors were of the mucosal type and six were of the intramural, extramucosal, variety. The latter group consisted of two leiomyomas, one cyst and three tumors that were of indeterminate type but probably were leiomyomas.

Benign tumor may arise at any point in the esophagus. In our group the lower third of the esophagus was the most frequently involved. The large, pedunculated tumors, however, were much more likely to arise from the upper end of the esophagus than from other levels. The tumors may be single or multiple, single tumors being twice as common as multiple. The mucosal tumors are generally sessile and frequently become pedunculated. This is due to the position of the tumor and to the fact that it is subjected to the peristaltic action of the esophagus, which tends to elongate and mold the tumor and give rise to a pedicle, which may vary in length and thickness. Through this action, a tumor originating in the upper end of the esophagus may extend down to the cardia or even through it and into the stomach. The dependent portion of the tumor is usually rounded and tapers toward the base but it may assume various shapes and may be bifid or lobulated. Tumors arising from the outer coats of the esophagus tend to escape the peristaltic pull and seldom become pedunculated. A benign tumor of the esophagus is usually covered with normal appearing mucosa, although areas of ulceration may appear as a result of pressure or trauma.

Patterson stated that benign tumor of the esophagus seldom produces symptoms. Barrett[4] expressed the opinion that benign tumors of the esophagus of the intramural, extramucosal type do not give rise as a rule to esophageal symptoms unless the tumor encircles the wall of the esophagus. Frequently, such a tumor remains silent until it reaches a size large enough to cause mediastinal pressure. While the 44 patients at whose necropsies benign tumor of the esophagus was found had not had any symptoms referable to the esophagus, this did not hold true in the 15 cases under consideration. If one includes regurgitation of a tumor into the mouth as an esophageal symptom, all the patients who had benign mucosal lesions had symptoms referable to the esophagus. Of the six patients who had intramural, extramucosal tumor, five patients had symptoms referable to the esophagus. In only one case out of the 15, therefore, was the tumor entirely silent. The situation of the tumor and the length of

TABLE 2.

CHARACTER OF FIFTEEN BENIGN TUMORS OF THE ESOPHAGUS FOUND AMONG 11,000 PATIENTS WHO HAD ESOPHAGEAL DISTURBANCE

CASE	TYPE OF TUMOR	AGE, YEARS	SEX	MUCOSAL OR EXTRA-MUCOSAL	LEVEL OF ORIGIN IN ESOPHAGUS
1	Lipoma	62	M	Mucosal	Upper third
2	Polyp	54	F	Mucosal	Middle third
3	Adenoma	48	M	Mucosal	Lower third
4	Hemangioma	71	M	Mucosal	Middle third
5	Myxofibroma	24	M	Mucosal	Upper third
6	Adenoma	64	M	Mucosal	Middle third
7	Lipoma	42	M	Mucosal	Upper third
8	Fibrolipoma	71	M	Mucosal	Upper third
9	Polyp	62	M	Mucosal	Lower third
10	Cyst	39	F	Extramucosal	Middle third
11	Myoma	56	F	Extramucosal	Lower third
12	Myoma	39	M	Extramucosal	Lower third
13	Indeterminate	61	F	Extramucosal	Middle third
14	Indeterminate	45	M	Extramucosal	Lower third
15	Indeterminate	42	M	Extramucosal	Lower third

its pedicle, if the tumor is pedunculated, are important factors in the production of symptoms.

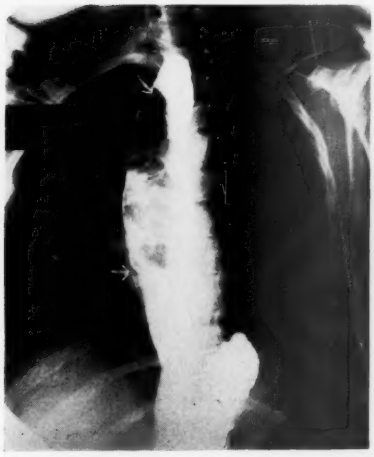
Arrowsmith[5] reported a small benign tumor of the esophagus with a very short pedicle situated at the esophagus introitus, which produced marked dysphagia and death. Rose reported a case of multiple small myomas in the wall of the esophagus which compressed the esophageal lumen entirely and produced death because of starvation.

Often, the first manifestation of a benign tumor of the esophagus is the sudden regurgitation of a fleshy mass into the mouth. It is fairly

common for an attending physician who has not had any experience with such tumors to doubt the patient's veracity and sanity if he does not see the patient while the regurgitated tumor is in the mouth. The patient invariably attempts in his panic to pull the tumor out of the mouth or to bite it off and, when this is not successful, to stuff the tumor back into the mouth and reswallow it. Asphyxiation of the patient due to blockage of the larynx may take place during the regurgitation of such a tumor.

Dysphagia is the most frequent symptom in cases of benign tumor of the esophagus. It may be sudden and severe in its onset or insidious in its development. It is often intermittent in type. The size that a benign tumor of the esophagus may attain without interfering with deglutition is remarkable. Regurgitation may be present at times and varies with the degree of esophageal obstruction. Loss of weight may occur and usually corresponds to the degree of dysphagia. Less frequently, the patient may pass blood by stool or emesis. If the tumor is large, and especially if it is of the intramural, extramucosal type, there may be a sense of substernal discomfort associated with cough and expectoration. Tumors arising in the lower end of the esophagus may give rise to a sense of epigastric discomfort.

The diagnosis of benign tumor of the esophagus can be made with ease when the patient presents a history of regurgitation of a fleshy tumorous mass into the mouth. In all other cases the diagnosis may be difficult and the condition may even escape detection until revealed at necropsy. Roentgenologic examination of the thorax may give the first clue to the presence of a benign tumor of the esophagus, by the presence of an abnormal mediastinal shadow. Roentgenologic examination of the esophagus is of great value in establishing a correct diagnosis. Schatzki and Hawes[6] have given an excellent description of the essential features in the roentgenologic diagnosis of benign tumor of the esophagus and its differentiation from extra-esophageal tumor pressing upon the esophagus. A review of the literature dealing with benign tumor of the esophagus and our own experience[7] reveals that the diagnosis cannot always be established correctly by roentgenologic examination. This applies even to tumors that are extremely large and that may fill the esophagus entirely. The error that is most frequently made in cases of a large tumor is confusion of the picture with that of cardiospasm. This error is attributable to the fact that the esophagus dilates with increase of the size of the tumor, and the tumorous mass itself has been misinterpreted as being due to retained secretion or food.



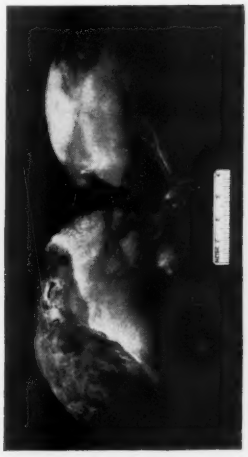


Fig. 1.—Roentgenogram showing an irregular intraluminal defect (between arrows). This is at the site of the multilobular lesion seen esophagoscopically.

Fig. 2.—Myxofibroma removed from esophagus in Case 5.

Esophagoscopy can be of considerable value in the diagnosis of benign tumor of the esophagus. However, contrary to the statement usually made in the literature dealing with the problem of benign tumor of the esophagus, the diagnosis cannot always be made on esophagoscopic examination. Barrett,[4] Haenisch,[8] Samson and Zelman,[9] and others have reported benign tumors even of large size that have escaped esophagoscopic detection. This error is attributable to the fact that the mucous membrane covering most benign tumors of the esophagus has the same appearance as the esophageal mucosa itself and is so interpreted. Difficulty is encountered especially in the esophagoscopic differentiation between intramural, extramucosal tumors and extra-esophageal tumors pressing upon the esophagus deforming it. It is not always possible to obtain a satisfactory specimen for biopsy from benign tumors of the esophagus, as they are often covered with normal mucous membrane or they may be covered by such a dense fibrous coat that it is difficult to obtain satisfactory tissue.

While benign tumors of the esophagus seldom undergo malignant change, this may occur, a fact which emphasizes the necessity for early and prompt treatment.

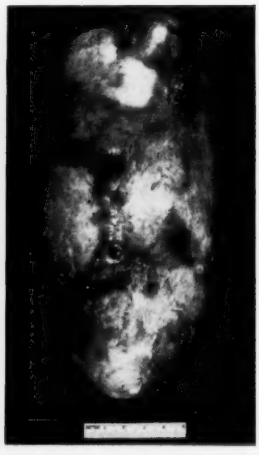


Fig. 3.—High-grade dilatation due to the large tumor of the esophagus associated with benign tumor, represented in Figure 4.

Fig. 4.—Large pedunculated lipoma removed from the esophagus in Case 7.

The treatment of benign tumor of the esophagus depends on the size of the tumor, its location, its character and the difficulty that it produces. If the tumor is small and does not produce symptoms it may not require treatment. The extramucosal, intramural tumors, if producing difficulty, are best handled surgically. If the tumor has a long pedicle, is attached about the esophageal introitus and is of such size that it can be brought out through the mouth, it usually can be removed, as suggested by Annandale[10] in 1878 and by Garretson and Hardie[11] in 1928, with a snare applied under esophagoscopic guidance and the pedicle can be severed with surgical diathermy. Care must be exercised to secure a firm grasp of the tumor at the time of its removal, to prevent its aspiration into the trachea. Dubois,[12] in 1818, reported such a complication with fatal results. In certain selected cases the use of radium and the destruction of the tumor by surgical diathermy may be feasible. Very large tumors and those not amenable to the forms of treatment outlined are best handled surgically.

REPORT OF CASES

A brief abstract is presented of data on the benign tumors of the esophagus included in our study. For purposes of classification they

are divided into the mucosal and the intramural, extramucosal tumors. Data on four of the mucosal tumors (Cases 1 to 4, Table 2) have been reported previously[2, 7, 13, 14] and the reports will not be repeated here.

CASE 5 (Table 2).—A man, 24 years of age, always enjoyed good health until three and a half years prior to our examination. At that time a sore throat had developed, which lasted two weeks. During the next three and a half years the patient had had six such spells. With the onset of the sore throat there was a tendency for mucus to accumulate in his throat and he would expectorate copious amounts of clear mucoid material. On two occasions the sputum had contained blood and on several occasions the patient had passed tar-colored stools. Soon after the onset of sore throat, dysphagia had developed. This had been highly variable. For a week or so the patient was able to eat everything without trouble and again the esophagus might close up completely for a day or two at a time. When the dysphagia was particularly severe there was usually an associated sense of substernal pressure. On several occasions during the three and a half years previous to our examination, the patient had regurgitated a large, fleshy mass out of the mouth. He had attempted to bite off the mass but had been unsuccessful and was forced to reswallow it. The last time that this occurred marked dyspnea and strangulation had developed and the patient had experienced considerable difficulty in reswallowing the tumor. Numerous roentgenologic and esophagoscopic examinations had been performed but the true nature of the difficulty had not been recognized.

The patient was admitted to the Mayo Clinic September 7, 1940. He was undernourished and had marked clubbing of the fingers. On percussion of the thorax a definite widening of the mediastinum was noted anteriorly. Roentgenologic examination of the esophagus was reported as revealing cardiopasm (Fig. 1). On esophagoscopy, a large, pedunculated and lobulated tumor was found, which was attached to the posterior wall of the esophagus just below the cricoid cartilage. The tumor extended down into the middle third of the esophagus. An attempt was made to bring the tumor out of the esophagus but it was so tightly wedged in the esophagus that this was impossible. Cervical esophagotomy was performed and a large tumor (Fig. 2) was removed, which proved to be a myxofibroma.

CASE 6.—The patient was a man 64 years of age. A year and a half before admission to the clinic, he had noticed gradually increasing dysphagia. At the time of our examination on December 30, 1937, his diet was restricted to liquids. In spite of the restricted

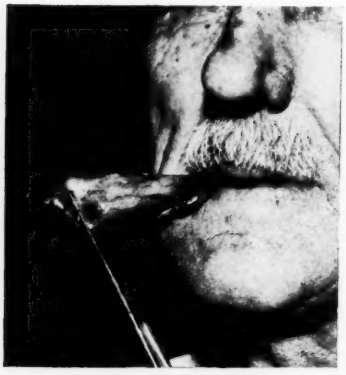


Fig. 5.—Benign pedunculated tumor of esophagus brought out of mouth in Case 8.

Fig. 6.—Roentgenogram showing an elongated defect in the upper part of the esophagus (arrows). The roentgenologic diagnosis was pedunculated tumor.

diet, there had not been any loss of weight. On esophagoscopy, a tumor was found 13 inches (33 cm.) from the incisor teeth. Tissue removed from the lesion was reported on microscopic examination as an adenomatous polyp. During the next two years, the patient's esophagus was dilated five times by means of Plummer sounds with temporary relief of symptoms.

CASE 7.—The patient, a man 42 years of age, came under our observation October 21, 1943. His chief complaint was that of pressure in his thorax, which had been present since 1935. Soon after the onset of his difficulty he had begun to regurgitate and vomit undigested food. Since 1939, the patient had vomited undigested food after practically every meal and as a result he had lost 30 pounds (14 kg.). During the past few years he had had difficulty sleeping at night in a recumbent position, as secretion tended to run into his mouth and cause strangulation. He had had to sleep in a chair for a year preceding our examination. Numerous roentgenograms of the esophagus had been made and esophagoscopy had been performed five times. A diagnosis of cardiospasm had been made. Many dilators had been passed without benefit.

Our roentgenologic examination of the esophagus revealed that it was much dilated but the gastric opening seemed sufficient to allow food to pass into the stomach. The true nature of the process was not recognized roentgenologically (Fig. 3). Esophagoscopy revealed a large, pedunculated, intra-esophageal tumor covered with normal esophageal mucosa. The tumor was attached by a pedicle to the upper end of the esophagus immediately below the cricoid level and extended down to the cardia. It was only after the tumor had been found on esophagoscopy that the patient recalled having regurgitated a fleshy mass into his mouth six years previously. He had attempted to bite the mass off but, being unsuccessful, had pushed it back into his throat.

Surgical treatment was advised. Because of the size of the tumor transpleural esophagotomy was deemed advisable. This was done and a tumor measuring 22 by 8.5 by 5 cm., without the pedicle, and weighing 570 gm. was removed. On microscopic examination it was found to be a polypoid lipoma (Fig. 4).

CASE 8.—A man 71 years of age was admitted to the Clinic March 1, 1944, with a history of having regurgitated a fleshy mass into his mouth. Ten or twelve years before admission the patient first had noticed, after a coughing spell, a lump on the back of his tongue. He attempted to cough this out without success and after

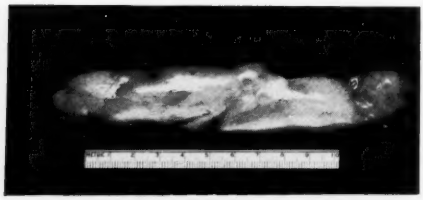


Fig. 7.—Pedunculated fibrolipoma removed in Case 8.

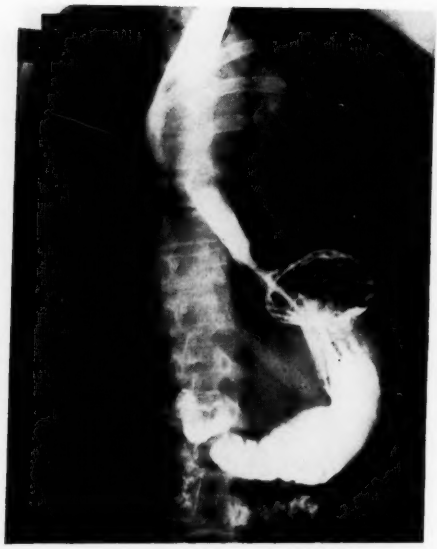


Fig. 8.—Intramural extramucosal tumor arising in the wall of esophagus.

a few moments it slipped away. Three weeks later the same thing occurred. Seven years ago, the patient again had regurgitated the mass and this time it reached as far as his teeth. Three years ago, after a vomiting spell, the tumor again had reappeared and projected out of the mouth. At this time he attempted to bite off the tumor without success and again swallowed it. The patient had been examined roentgenographically and an esophagoscopy had been performed. A diagnosis of a benign pedunculated tumor of the esophagus had been made and surgical treatment had been advised but the patient had refused the advice.

On our examination, the diagnosis of a pedunculated, benign, esophageal tumor was confirmed. Roentgenologic examination of the esophagus demonstrated the lesion, which appeared attached to the left wall of the esophagus (Fig. 6). On esophagoscopy a pedunculated tumor was found in the esophagus. The tumor extended down into the middle third of the esophagus and was attached by a pedicle to the left lateral wall of the esophagus just below the level of the cricoid cartilage. The lower end of the tumor was grasped with forceps and the tumor was brought out of the mouth (Fig. 5). A snare was slipped over the tumor and, under esophagoscopic guidance, placed about the base of the pedicle. The pedicle was then severed with a cutting current in order to prevent bleeding. The patient made a rapid and uneventful recovery. Microscopic examination revealed the tumor to be a pedunculated fibrolipoma (Fig. 7).

CASE 9.—The patient was a man 62 years of age, who gave a 25 year history of dysphagia that had increased slowly in severity. A year before admission to our care, he had been able to get very little food down and a gastrostomy had been performed. On admission to the clinic in April, 1928, a roentgenogram of the esophagus revealed a large tumorous mass in the lower part of the esophagus, which almost completely occluded the lumen. On esophagoscopy a large, polypoid tumorous mass, which had the appearance of multiple polyps, was found in the lower part of the esophagus. Numerous specimens for biopsy were removed from the mass and all were reported to have been the site of inflammation. Radium was implanted into the mass without any particular benefit to the patient.

There were six intramural, extramucosal tumors. In the cases in which the tumors were extramucosal the clinical picture differed somewhat from that in the cases in which the tumors were mucosal. Data on the cases are summarized briefly.

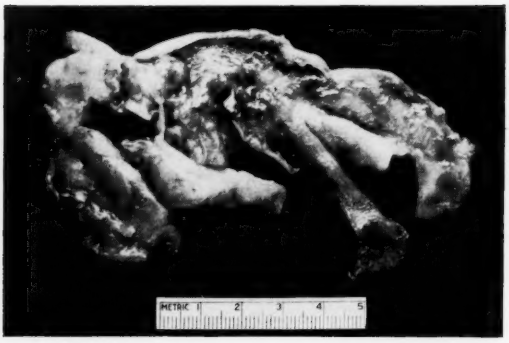


Fig. 9.—Cyst removed from wall of esophagus in Case 10.

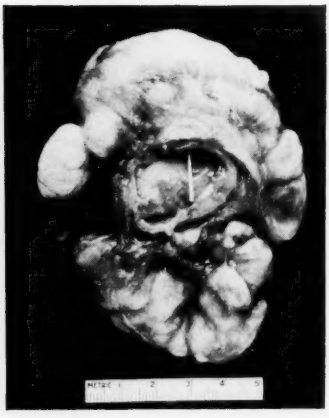


Fig. 10.—Leiomyoma removed in Case 11.

Fig. 11.—Cross section through leiomyoma and resected portion of esophagus.

CASE 10.—The patient, a woman aged 39 years, was first seen at the clinic March 8, 1943. She gave a history of cough for the past 15 years. For three months preceding admission she had raised small amounts of mucoid secretion. Recently, she had noted a diffuse substernal pain on swallowing.

Roentgenologic examination of the thorax revealed a large, rounded, soft tissue mass measuring approximately 7 cm. in diameter in the posterior mediastinum at the level of the seventh to the tenth thoracic vertebrae. The tumor appeared to spread the main stem bronchi apart. On roentgenologic examination of the esophagus a tumor measuring 5 by 8 cm. was found lying posteriorly and to the right side of the middle third of the esophagus, compressing the esophageal lumen. It appeared to arise in the wall of the esophagus (Fig. 8). On bronchoscopy, the coryna was found markedly flattened. The right main stem bronchus was displaced laterally and the lumen was reduced approximately to half its normal diameter owing to pressure on the medial wall. The left main stem bronchus was displaced further to the left than normal. On esophagoscopy the esophagus was found to be displaced to the right by a mass at the level of the middle third of the esophagus but it was impossible to say whether the displacement was due to an extra-esophageal or an intra-esophageal tumor. At operation, a cystic tumor was found involving the wall of the middle third of the esophagus (Fig. 9). The cyst was dissected out of the wall of the esophagus and was found to contain six ounces (170 gm.) of thick, creamy material. Microscopic examination was reported as showing an infected cyst lined with glandular epithelium and containing smooth muscle in its wall. The patient made a satisfactory recovery.

CASE 11.—The patient was a woman 56 years of age, who was referred to the clinic May 17, 1943, with the diagnosis of a tumor, probably a leiomyoma, involving the lower end of the esophagus and the cardiac end of the stomach. She had been well until a month previous to admission, when a dull pain beneath the xiphoid process and an occasional sense of solid food lodging at that point had developed.

Roentgenologic examination of the thorax showed a tumorous mass lying to the right of and behind the distal portion of the esophagus. Examination of the esophagus revealed an obstruction at its lower end due to a tumor lying to the right of and behind the distal portion of the esophagus and involving the right aspect of the cardiac end of the stomach, extending apparently through the esophageal hiatus. The tumor appeared to be intramural. Esophagoscopic ex-

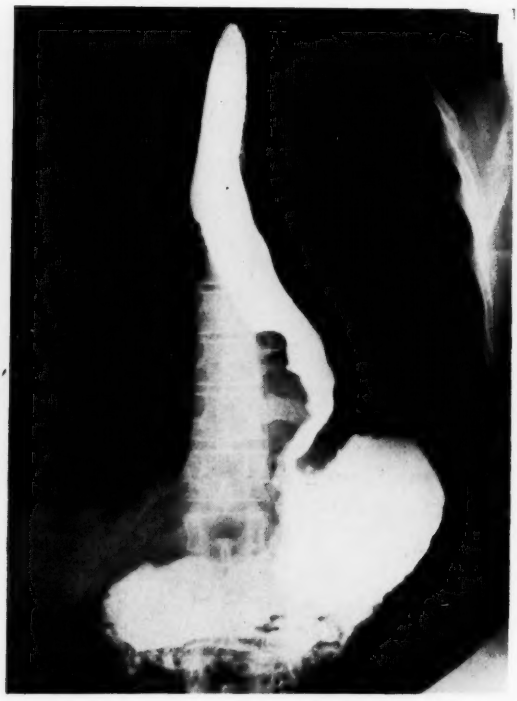


Fig. 12.—Leiomyoma involving lower end of esophagus in Case 12.

amination confirmed the roentgenologic findings and a tentative diagnosis of leiomyoma involving the lower part of the esophagus and the cardiac end of the stomach was made.

On abdominal exploration, a large tumor was found involving the posterior wall of the cardiac end of the stomach and the lower end of the esophagus. Only the abdominal portion of the tumor was removed and proved to be a leiomyoma. The portion removed measured 15 by 5 by 5 cm. (Fig. 10). The patient made a satisfactory recovery.

CASE 12.—The patient, a man 39 years of age, was first seen at the clinic May 18, 1942. His chief complaints were dizziness and asthma. On roentgenologic examination of the thorax, a shadow was noted behind the left border of the heart. Roentgenologic examina-

Fig. 13.—Intramucosal tumor of esophagus (arrows).

tion of the esophagus revealed a mass intrinsically associated with the wall of the esophagus, which was considered as a possible leiomyoma (Fig. 12). Esophagoscopy revealed a marked deformity of the lower end of the esophagus due to pressure of the tumor. It was impossible to determine the character of the lesion. Exploration was performed and a tumor was found, which arose in the lower end of the esophagus and completely surrounded and compressed its lumen. The lower end of the esophagus was resected and the tumor was found to be a leiomyoma (Fig. 11).

CASE 13.—The patient, a woman aged 61 years, was seen first at the clinic February 26, 1944. He chief complaints were retrosternal pain and dyspnea. The pain had been present three years and was aggravated by exertion, movement and eating. Roentgenologic

examination of the esophagus showed a filling defect in its anterior wall at the level of the sixth thoracic vertebra. A diagnosis of intramural esophageal tumor was made (Fig. 13). On esophagoscopy, a tumor was found involving the left wall of the esophagus. It was covered with normal mucosa and had the appearance of a myoma. The patient's pain was thought to be due to angina pectoris rather than to the esophageal tumor.

CASE 14.—The patient, a man 45 years of age, had had intermittent spells dysphagia for the past seven years. On roentgenologic examination of the esophagus, a lesion was found in its lower end. On esophagoscopy, a tumor was found in the wall of the esophagus just above the cardia. The appearance of the tumor was suggestive of a leiomyoma. It is of interest that an operation had been performed seven years previously because of gastro-intestinal bleeding and at the time of operation numerous small leiomyomas had been found scattered over the first 18 inches (46 cm.) of the jejunum and the antral portion of the stomach.

CASE 15.—The patient was a man 42 years of age. For the past four or five years he had had gastric distress when lying on his back. Two or three years prior to admission, he had first noted that food would stick in the esophagus and that this would be relieved by vomiting. The dysphagia had been intermittent. Roentgenologic examination revealed a tumor in the lower part of the esophagus. The tumor appeared benign. On esophagoscopy, a tumor two inches (5 cm.) in length was found to involve the left lateral wall of the esophagus. The lesion looked like a leiomyoma. The patient is still living five years after his last examination.

CONCLUSIONS

1. Benign tumors of the esophagus are comparatively rare and are characterized by the fact that they reach considerable size without production of symptoms.

2. They may be divided into two types, those arising from the mucosa or submucosa and those arising from the outer coats of the esophagus. The latter are often called intramural, extramucosal tumors. The mucosal tumors are more prone to give rise to dysphagia than are those arising from the outer coats of the esophagus.

3. Benign tumors of the esophagus are found more frequently among men than among women and although found at all ages they are encountered most frequently after the fourth decade.

4. The diagnosis of benign tumor of the esophagus is relatively simple if the patient gives a history of regurgitation of a fleshy mass. In all other cases, considerable difficulty may be experienced in arriving at a correct diagnosis.

5. Correct diagnosis of benign tumor of the esophagus is important, for such tumors are generally amenable to treatment.

MAYO CLINIC.

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LXXXV

OBSTRUCTION OF THE RIGHT MAIN BRONCHUS DUE TO CONGENITAL MALDEVELOPMENT OF THE PULMONARY VEINS

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The patient here reported was admitted to Babies' Hospital July 9, 1943, at the age of four months. His birth had been normal; his birth weight was over 11 pounds. Some respiratory difficulty was evident even at birth, and his mother was advised to make him cry in order to expand his lungs. It was noted that he had unusually rapid respiration.

He did fairly well until he was three months old, although he gained little weight. At that time, about six weeks before admission to the hospital, cyanosis was observed for the first time, and his loud cry had disappeared. He had, moreover, some difficulty in finishing his feedings without resting.

On the day of admission to the hospital he had an attack of cyanosis of such severity that his life was despaired of, and because of this he was admitted to the hospital. On admission the baby was extremely uncomfortable, of a bluish-gray color, with extremely rapid respiration (96) and a pulse rate of 100. He could make no sound when he attempted to cry. There was abdominal and supersternal retraction on respiration.

The heart appeared to be greatly enlarged; the right lung was emphysematous, the left compressed. A murmur of uncertain origin could be heard over the whole chest.

Films of the chest were reported to show the following: The superior mediastinum is widened, particularly on the right side. The trachea is buckled at the level of the clavicle, the heart appears enlarged.

The symptoms to be explained were the attacks of cyanosis with rapid respiration and pulse, the loss of voice, and the widened mediastinal shadows.

Fig. 1.—The greatly enlarged heart consisted chiefly of dilated and hypertrophied right auricle and right ventricle. The pulmonary artery was dilated and the aorta was small. The superior and inferior venae cavae entered normally into the right auricle. The proximal part of the superior vena cava and the right innominate vein were greatly enlarged, resulting in a fusiform venous dilatation, partially covered anteriorly by the right lobe of the thymus. These dilated veins corresponded in location with the supracardiac shadow seen in roentgenograms. The left innominate vein was large also and a persistent left vena cava coursed downward anterior to the left pulmonary artery and behind the heart.

The diagnoses suggested were congenital heart disease, thymic tumor, tracheal or laryngeal pathology, anamolies of the great vessels.

In the hospital the baby continued to have attacks of severe cyanosis and dyspnea with supraclavicular and intracostal retraction. These were of such a nature that it was felt that there must be either a laryngeal obstruction, or external pressure on the trachea. Accordingly a tracheotomy was done on July 29, three weeks after admission. This resulted in no improvement. The next day a bronchoscope was passed through the tracheotomy opening and a tumor was seen anteriorly compressing both primary bronchi. It was soft, and the tube could easily be pushed beyond it. This resulted in improve-

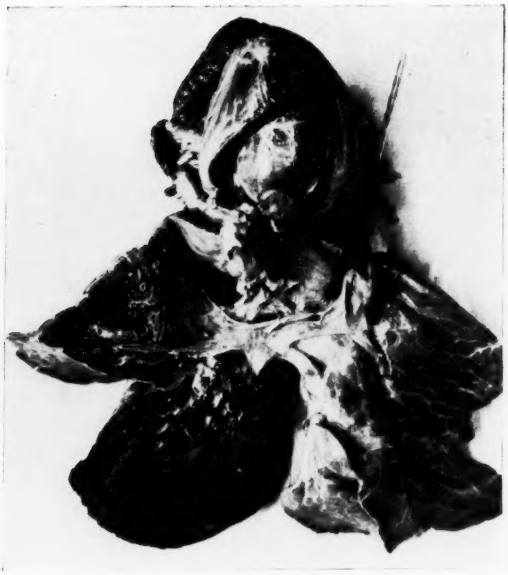

Fig. 2.—Taken with the heart reflected. The left superior vena cava failed to enter the pericardial sac and the coronary sinus, but was continuous with a large venous trunk which ran horizontally toward the right lung behind the ascending aorta and pulmonary artery, just below the level of the bifurcation of the trachea. This horizontal trunk originated from the union of the veins of the upper and middle lobes of the right lung. Midway in its course it received large veins from both lower lobes and at the angle at which it joined the left superior vena cava, veins from the left upper lobe entered.

ment of the air exchange volume while the tube was in, but no change in the respiratory rate. It was noted that the larynx was normal. Since the loss of voice could not be explained by anything in the larynx, it was concluded that the expiratory air column was so feeble as to be insufficient to produce any sound in the larynx. The bronchoscopy demonstrated a tumor in the chest but gave no hint as to its nature.

After the bronchoscopy the tracheotomy tube was removed. The baby continued to have attacks of cyanosis, gradually became weaker and died August 6, one month after admission.

Fig. 3.—As a result of the venous anomalies the entire return of blood from the lungs to the heart took place through these anomalous pulmonary veins, persistent left superior vena cava, left innominate vein and right superior vena cava to the right auricle. Into this auricle entered also the systemic venous return. This photograph shows the resulting dilatation of the right auricle and ventricle and a patent foramen ovale, the sole communication between the hypertrophied right cardiac chambers and the small left auricle and ventricle and hypoplastic aorta.

The autopsy explained the clinical appearances. This was done by Dr. Paige, who allows me to use her report. For our purposes it is necessary to give only what concerns the heart and great vessels. I summarize the report of Dr. Paige.

"The heart is large measuring 8.5 cm. in transverse diameter. Its left margin extends to the left lateral chest wall. The left lung is markedly compressed behind the heart. The right lung is expanded. The thymus measures 4.5 cm. in transverse diameter. The right lobe of the thymus partially covers a mass which projects beyond its right border. This mass proves to be the greatly enlarged superior vena cava and the right innominate vein, the dilatation occuring in the vicinity of the entrance of the left innominate vein. On the medial surface of the upper lobe of the right lung is a depression in which the dilated veins lie. The greatly enlarged heart consists chiefly of the right auricle and ventricle. The right auricle is dilated and hypertrophied and its endocardium is thickened and white, but smooth. It receives the superior and inferior venae cavae. The fora-

men ovale is patent along its anterior margin, an oval opening measuring 11 by 8 mm. The coronary sinus opens in normal position. The right ventricle is also dilated and hypertrophied, especially in the conus region, and much of its endocardium is slightly thick and gray.

"The left auricle and ventricle are small. No pulmonary veins enter the left auricle. The inferior and superior venae cavae enter the right auricle in the usual manner. The latter measures 1.3 cm. in diameter for a distance of 1.3 cm. and cephalad to this region it dilates to 2 cm. in width where the left innominate vein joins it. The dilatation continues into the right innominate vein and results in a roughly spindle-shaped venous dilatation 3.5 cm. in length. The left innominate vein is large and measures 8.5 mm. in width. Nine mm. proximal to the union of the left internal jugular and left subclavian veins a large tributary enters the left innominate vein. This measures 1 cm. in diameter and 6.5 cm. in length. It passes downward anterior to the left pulmonary artery and then turns sharply to the right and courses just below the bifurcation of the trachea toward the right lung. In its course this anomalous vein receives tributaries from all lobes of both right and left lungs, the entire venous pulmonary return taking place through the large anomalous vein, left innominate vein and superior vena cava to the right auricle."

These findings completely explain the clinical symptoms. The appearance of a mediastinal tumor was caused by the greatly dilated right superior vena cava. The dilatation was caused by the pulmonary venous return brought to it by a persisting left superior vena cava which received all the pulmonary veins.

A persisting left superior vena cava receiving the pulmonary veins is one of the rarest anomalies of the great vessels in the mediastinum. The mere presence of a left superior vena cava is by no means very rare and if it joins the right side of the heart it may cause no symptoms. If, however, it receives the pulmonary circulation and then joins the right side of the heart, the outcome is sure to be fatal.

One would say that in this case the congenital heart disease, that is, the open foramen ovale, had little to do with the symptoms; in fact, it was rather a life-saving anomaly since only through it could any blood reach the left heart and the general circulation.

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LXXXVI

SPONTANEOUS MEDIASTINAL EMPHYSEMA REPORT OF A CASE

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Spontaneous mediastinal emphysema is a clinical entity first described by Hamman in 1937 and again in 1939.[1, 2] Hamman was greatly puzzled by the first case which he encountered and failed to make a diagnosis until he observed a second case with more characteristic symptoms and physical findings. Subsequently he was able to collect five additional cases. Since this original report, cases have been recorded by McGuire and Bean,[3] Morey and Sosman,[4] Monroe and Webb,[5] Gumbiner and Cutler,[6] and by Fisher,[7] and others. The cases of the last three mentioned authors occurred in newborn infants.

The symptoms usually appear suddenly in a previously healthy individual who gives no history of any unusual precedent exertion or of trauma of any kind. The patient complains of a more or less severe pain in the region of the precordium from which it may radiate into the neck and arm. There may be a feeling of tightness in the chest with shortness of breath on exertion. Subsequently, a soft crepitant swelling may be noticed at the root of the neck anteriorly and may spread onto the face and the chest.

On physical examination the pulse and respiratory rate may be increased but in adults there is usually no febrile reaction. In infants and children, fever, dyspnea and cyanosis may occur. On examination the heart sounds may appear muffled and distant. A peculiar "crunching," "crackling" sound synchronous with the heart beat may be heard over the precordium. The sound is best elicited when the patient lies on the left side. Subsequently a crepitant subcutaneous swelling may appear above the sternal notch from which it may spread onto the face and the chest. The roentgenograms show some widening of the mediastinal shadow and the lateral view reveals the presence of air bubbles in the anterior, superior and posterior med-

*Published posthumously.

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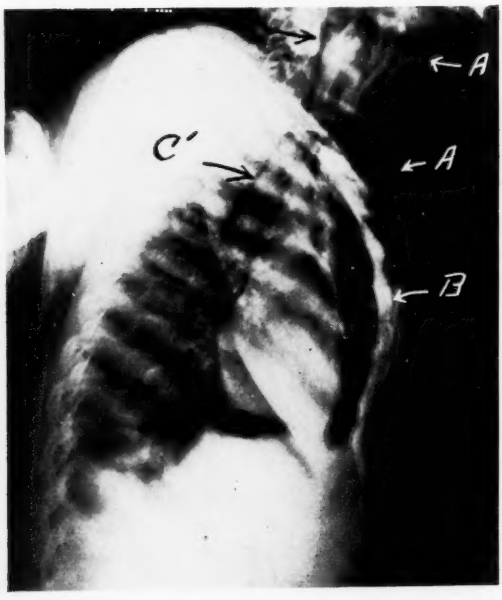

Fig. 1.—Showing the presence of air in the subcutaneous tissues of the neck and chest, A-A'; in the anterior mediastinum at B and in the areolar tissues about and behind the pharynx and the esophagus, C-C'.

iastinum. This triad, together with the history, usually suffices to establish the diagnosis.

The severity of the symptoms will vary according to the degree of tension established by the air confined within the mediastinum—increasing tension interfering more and more with the return flow of blood to the heart.

The following case represents a striking example of spontaneous mediastinal emphysema.

REPORT OF A CASE

M. S., a three-year-old colored female was brought to the Cincinnati General Hospital by her mother shortly after noon on December 6, 1942. The mother stated that the child had had a slight cold the night before and had developed a moderate cough at four o'clock in the morning. Six hours later, while in the mother's lap,

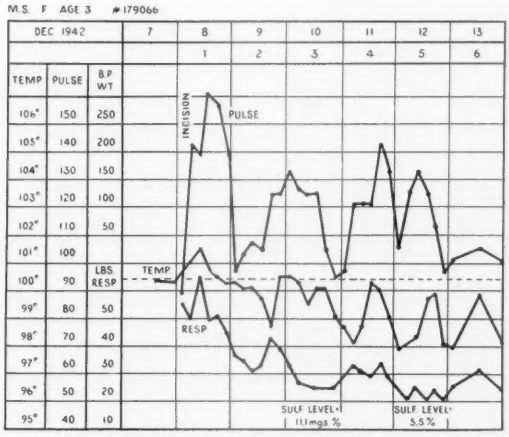

Fig. 2.—Chart showing marked increase in the respiratory and pulse rates with but slight elevation in the temperature.

a swelling had been noticed in the midline of the child's neck just above the sternoclavicular articulations. The swelling began to spread up the neck onto the face and into the left upper eyelid. There had been considerable difficulty in breathing since the coughing spell earlier in the day. The child had previously enjoyed excellent health aside from thrush and whooping cough in 1939 and measles in 1940.

On admission to the hospital the temperature was 100° F. rectal, the respiratory rate 60, and the pulse rate 150. The well-developed child was in respiratory distress and appeared acutely ill. There was a swelling in the front of the neck extending onto the left cheek and into the left eyelid and also extending downward over the left chest anteriorly. Crepitus was present in all these areas. The tonsils were markedly enlarged and reddened with some exudate present. The posterior wall of the pharynx was swollen as far as the hypopharynx, probably because of the air bubbles. The heart sounds were clear and the rate and rhythm were rapid. No murmur was heard and no crunching sound was heard over the precordium. Examination of the lungs disclosed that the percussion note was slightly hyperresonant with loud expiratory brassy sounds associated with many medium and coarse râles in all lung fields. The respiratory rate was irregular.

Roentgenograms of the chest showed the presence of a large amount of air in the areolar tissues in front of the heart and also in the superior and posterior mediastinum pushing the pharynx and the esophagus away from the vertebral column (Fig. 1).

The white blood count was 22,000. The remaining findings were noncontributory.

A diagnosis of spontaneous mediastinal emphysema was made but it was deemed necessary to rule out an aspirated foreign body.

On the evening of the same day bronchoscopy was performed with local, 1% pontocaine anesthesia. The tracheobronchial tree was negative and no foreign body was found. An incision was then made in the midline of the neck from the lower border of the cricoid ring to the sternal notch. Air was encountered and escaped from the wound when the subcutaneous tissues were incised. The pretracheal fascia was then incised but apparently no air escaped from beneath the fascia. The wound was left open.

The patient was placed in an oxygen tent and sulfathiazole medication was initiated.

On the two succeeding days there was no improvement in the patient's condition and there was no decrease in the amount of the subcutaneous emphysema. By the fourth postoperative day the subcutaneous and mediastinal emphysema, visible by x-ray, began to diminish and the general condition improved. The white count came down to 12,250. From then on the patient made an uninterrupted recovery and was discharged from the hospital 12 days after the operation (Fig. 2).

DISCUSSION

The experimental work on animals by Macklin,[8, 9] elucidates the pathogenesis of spontaneous mediastinal emphysema. Under varying degrees of pressure Macklin forced air into the bronchi of cats and other animals and was able to produce a mediastinal emphysema. At autopsy the pathway of the air could be traced from the alveoli (ruptured) along the sheaths of the pulmonic vessels into the mediastinum. The air accumulated within the connective tissue sheaths of the vessels and tended to compress the latter. In the mediastinum the same phenomenon was noted about the larger vessels, especially about the veins entering both sides of the heart. The pneumomediastinum thus produced accounted for the cyanosis, dyspnea and heart failure which had occurred in the animals before death. Ballon[10] distended the

mediastinum of rabbits with free air or with inflated balloons. Following these experiments it was noted that with each injection of air there was a fall in blood pressure and an increase in the respiratory rate. "Only gradual rises and falls in venous pressure followed no definite rule. Transitory terminal rises concomitant with the fall in arterial pressure could be observed."

Aside from the characteristic signs and symptoms mentioned above, spontaneous mediastinal emphysema must be differentiated from "surgical" mediastinal emphysema following trauma to the chest either from without or within, and including foreign bodies in the bronchi or the esophagus. In the newborn other causes of asphyxia must be ruled out. Angina pectoris, coronary occlusion, pericarditis, pleurisy, pneumonia, spontaneous pneumothorax and pulmonary infarction must be considered.

The prognosis in uncomplicated cases without tension pneumomediastinum is good. In the latter condition surgical intervention is often indicated.[11]

In uncomplicated cases expectant treatment will usually suffice and the air will be absorbed spontaneously. In cases with tension, cervical mediastinotomy either by a midline or transverse incision is indicated and with either approach the pretracheal fascia should be opened. In the newborn, in a few instances, relief has been obtained by puncture and aspiration of the anterior mediastinum.

SUMMARY

A brief review of the signs and symptoms and of the pathogenesis of spontaneous mediastinal emphysema is given. A severe case of this kind, occurring in a three-year-old child, is reported. The surgical and medical treatment which was employed, and which lead to the recovery of the patient is described.

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LXXXVII

EMERGENCY CERVICAL MEDIASTINOTOMY IN A CASE OF MASSIVE MEDIASTINAL AND SUBCUTANEOUS EMPHYSEMA SECONDARY TO REMOVAL OF A FOREIGN BODY FROM THE BRONCHUS

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Various cases have been reported of subcutaneous, interstitial and mediastinal emphysema in the literature. Most commonly, the emphysema resulted from violent respiratory efforts as seen in obstetric labor, obstructive tracheobronchitis, tracheotomy and pertussis; in other cases it followed traumatic chest injury, thyroidectomy, tonsillectomy, pneumonia and influenza.

Extensive subcutaneous and mediastinal emphysema secondary to the aspiration of foreign bodies into the tracheobronchial tree has been observed by Forbes,[1] and Vinson and Moersch.[2] Forbes in 1926 noted marked subcutaneous emphysema involving the neck and chest in a child following the aspiration of a peanut into the lower right lung three days previously. Vinson and Moersch published a report of three cases in 1931. In the first, a 6-year-old boy had aspirated a metal paper clip into the right bronchus. Twenty-one hours later subcutaneous emphysema was present over the anterior thorax and neck on the right side. In another, 11 days after the aspiration of a peanut into the right main bronchus, marked swelling of the head and neck developed following a severe coughing spell. X-ray examination showed emphysema of the upper portion of the right lung with atelectasis of the lower portion of the lung and the presence of air in the pericardial space. There was also pronounced emphysema of the tissues of the head, neck, upper portion of the thorax and of the arms. In a third case, emphysema of the upper thorax and neck followed aspiration of a peanut and gradually increased with each spell of coughing. The bronchoscopic removal of the foreign bodies in all these cases led to complete recovery.

That mediastinal emphysema can assume a more dangerous course was observed by Fisher and Machlin[3] in cases of tracheobronchial obstruction. These authors reported the death of a 22-month-

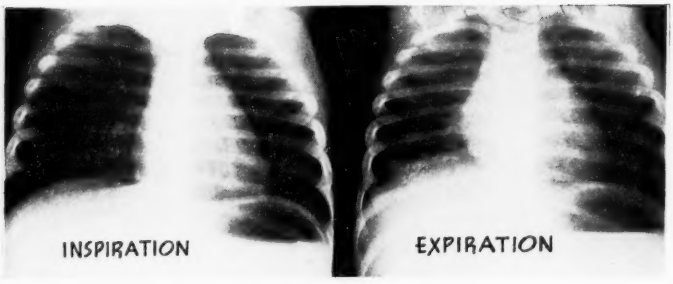

Fig. 1.—Roentgenograms illustrating obstructive emphysema caused by a piece of apple in the left main bronchus. Note the shifting of the heart and mediastinum to the affected side on inspiration and to the opposite side on expiration.

old infant following the aspiration of pieces of peanut, death being due to circulatory embarrassment and pressure from the emphysematous air. Michels[4] noted a 50 per cent mortality in six cases of pneumothorax and mediastinal emphysema following tracheotomy.

When signs and symptoms of progressive mediastinal emphysema occur, immediaate operation often becomes imperative as a life-saving measure. Such a situation arose in the case which is described.

REPORT OF A CASE

L. R. G., an 18-month-old, white, male child was admitted to Harper Hospital on January 8, 1944, because of a suspected aspirated foreign body. One and one-half hours previously the patient had choked on a piece of apple with resultant cough and asthmatic wheeze. Dr. George Moriarity was called and he advised a roentgen study of the chest. Fluoroscopy and roentgenography by Dr. V. C. Johnson revealed a typical check valve obstruction in the left main bronchus, probably due to a nonopaque foreign body. (Fig. 1.)

Physical examination showed a normal appearing child in no apparent respiratory distress. An audible expiratory wheeze was noted without the aid of the stethoscope. The left chest was tympanitic with diminished breath sounds. Bronchoscopy was advised at once. Without anesthesia, a 5 mm. bronchoscope was introduced. A foreign body was found in the orifice of the left main bronchus. A piece of the foreign body was removed with forceps, following

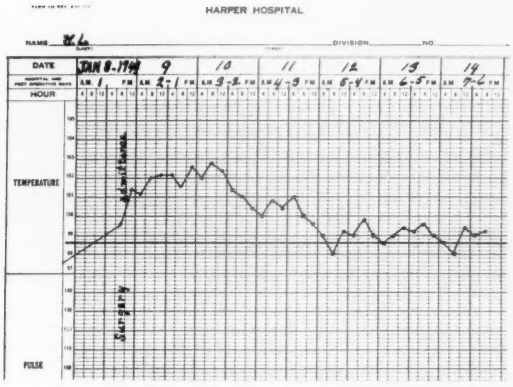

Fig. 2.—Temperature chart illustrating acute reaction of the body tissues to the emphysema.

which the remaining portion was suddenly and forcibly expelled through the bronchoscope by a forced expiration. The foreign body was a piece of apple. It was noted that the child struggled considerably during bronchoscopy but appeared to be in good condition upon leaving the operating room. No pulmonary distress or emphysema was noted.

Twenty minutes later the author was urgently called to the pediatric floor to see the patient. The child had been returned from the operating room crying continuously. Swelling of the tissues of the neck and the upper chest was first noted by the nurse, following which respiration became labored and the abdomen began to swell. The picture was shocking; the whole body was emphysematous, particularly the abdomen which had the appearance of a balloon which was rapidly becoming over-inflated with each respiration. The eyelids were bulging and the scrotum was as large as two adult fists. Crepitation was noted over the entire body extending to the fingers and toes. Dr. Leonard Gaydos, House Officer, who first saw the patient on the floor made the following notation on the chart: "The entire body had the feeling of a soft, thickly padded rug, there being approximately a two-inch cushion of air between the scalp and the skull."

Obviously the child's condition was critical. Prompt relief from this inflationary process was necessary if the patient was to survive.

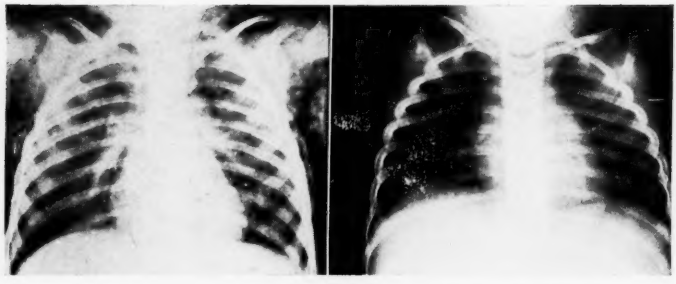

Fig. 3.—Left, portable roentgenogram taken of the chest on the first postoperative day. Extensive subcutaneous emphysema extending into each axillary area and into the supraclavicular region is present. No evidence of any pneumothorax or atelectasis is observed. Right, roentgenogram of the chest on the fourteenth postoperative day shows both lung fields to be normally aerated and the mediastinum in normal position.

The nurse, returning the baby to the operating room, made the remark that with each breath the baby became lighter and lighter and seemed to be floating away. Upon reaching the operating room the respirations were very labored and there was marked cyanosis. Immediate cervical mediastinotomy was decided upon. With a limited amount of local anesthesia, a midline incision was made from the thyroid notch to the sternum. There was a sudden gush of air from the subcutaneous tissues. Dissection was carried down to the trachea and laterally to the vascular sheaths. Finger dissection of the visceral and vascular fascia was carried down below the sternum with continuous release of air. Abdominal tension was relieved by multiple needle punctures under the skin. Within a few minutes, respirations appeared normal and the wound was lightly packed with vaseline iodoform gauze and the patient returned to bed.

The postoperative treatment consisted of a steam croup tent, sulfamerazine therapy, approximately one grain per pound of weight, and forced fluids. Convalescence remained indecisive for two days, then the temperature gradually subsided from 102.8° F. to normal on the sixth day. (Fig. 2) A slight temporary elevation occurred on the tenth and fourteenth postoperative days when the wound was prematurely closed with adhesive tape.

A portable x-ray study of the chest was made on the first postoperative day. This showed considerable subcutaneous emphysema, but there was no evidence of any pneumothorax or atelectasis. (Fig.

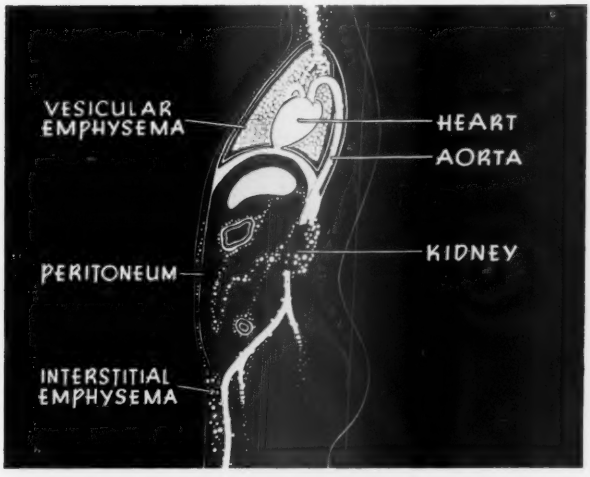

Fig. 4.—Emphysematous paths in relation to the pleurae, pericardium and peritoneum.

3) Shortly afterward, the air was aspirated from the scrotum with good results. Early on the second postoperative day, however, tension again became apparent over the abdomen. Deflation was accomplished at this time with a No. 16 gauge needle, resulting in prompt relief. The croup tent was discontinued on the fourth day and sulfamerazine therapy stopped on the eighth day. The emphysema gradually disappeared and the patient was discharged from the hospital on the sixteenth postoperative day with no clinical evidence of subcutaneous emphysema. (Fig. 3) The neck wound was not completely healed until March 1, 1944.

Experimental Data.—In 1919, Berkley and Coffen[5] observed extensive subcutaneous and interstitial emphysema complicating influenza and bronchopneumonia. Their autopsies showed that air gained access to the mediastinum from the lung without passing through the pleura. Rupture of a distended air sac and dissection of the air from the sac back to the hilus occurred. Streaks of air were seen leading off from the sacs, which followed the course of the large vessels.

Kelman,[6] in the same year at the University of Iowa, experimenting on rabbits found the path taken by air escaping from the

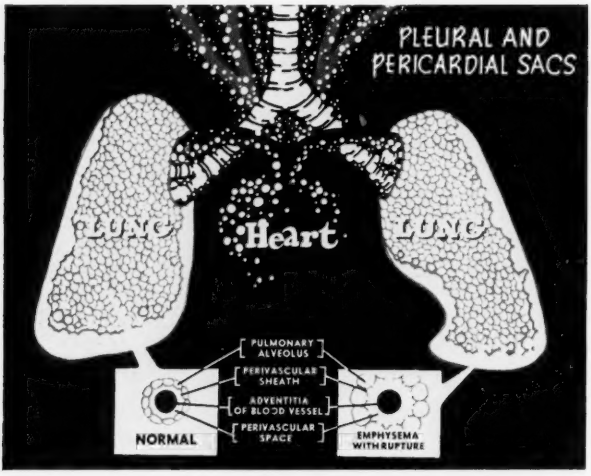

Fig. 5.—Relation of pleura, pericardium and blood vessels illustrating the direct path of the early interstitial emphysema in pericardial fat tissue (after Kelman) as well as Machlin's theory of rupture of the bases of the dilated air sacs into the perivascular sheaths of the smaller blood vessels.

lung into the tissues is by way of the root, following the reflexion of the pleura and the pericardium along the great vessels. (Fig. 4)

Ballon and Frances[7] at Washington University produced experimental emphysema in rabbits through tracheotomy and by varying the intratracheal pressure. They observed that the spread of air from the lung to the mediastinum occurred in streaks along the blood vessels.

In recent years, Machlin[8, 9] at the University of Western Ontario, has done extensive experimental work on cats on the same subject. He noted that the air breaks through the weakened bases of the alveoli which overlie the smaller branches of the pulmonary arteries and veins into the connective tissue around these vessels and from there, it makes its way along the vascular sheaths to the mediastinum. (Fig. 5) As the air accumulates, it presses on the vascular system, so impeding the circulation and possibly giving rise to pain as well.

Treatment.—The great majority of cases of emphysema respond to conservative care with rest in bed and control of cough and strain. In more severe cases, treatment must vary with the cause and progress

of the condition. If tension pneumothorax develops, it should be relieved. In rare instances, especially when life is threatened, the air may be removed by an incision at the root of the neck. To illustrate the various methods of operative procedure in cases of emergency, a brief review of the following five cases collected from the literature may be of interest.

CASE 1—1911—Tiegel[10] performed an emergency cervical mediastinotomy with a 4-cm. long vertical incision in the jugulum for a case of severe mediastinal emphysema in an adult following traumatic fracture of the ribs several hours previously. He applied a glass bell aspirator to the wound, which was connected to a water pump with a pressure of 30 cm. of water. The result was excellent.

CASE 2—1928—J. D. and P. C. Morton[11] reported a case of traumatic emphysema in a boy with a deep lung injury who developed severe subcutaneous emphysema over the head, neck and trunk within a few minutes after injury. Forty-eight hours later mediastinal tension was severe. Relief was obtained by two incisions parallel with the ribs on each side of the chest anteriorly, with the escape of a very considerable amount of air and an immediate improvement of symptoms.

CASE 3—1930—Meade and Stafford[12] observed spontaneous interstitial emphysema in a case of tuberculosis with an apical cavity in the left lung. For a period of 13 days, the dyspnea had been relieved by needle aspiration of air from the pleural cavity, by aspiration of air from the soft tissues and finally by short incisions made into the emphysematous tissues under each clavicle. On the fourteenth day, after all of these methods had failed to produce a final result, incisions were made in the supraclavicular regions with prompt and permanent relief of the dyspnea.

CASE 4—1938—Levent and Kohn[13] reported the case of a 2½-year-old child who developed emphysema secondary to an acute respiratory infection associated with coughing and violent efforts to vomit. Emphysema, extending over the neck, face and thorax, developed within a period of hours. An emergency opening of the mediastinum with the incision at the base of the sternum, and with finger dissection to the mediastinum, became an imperative procedure. The authors were first disappointed not to have seen the air bubbles come out, or as they say, "Nous avons à ce moment, une petite désillusion de ne pas voir apparaitre les bulles d'air que nous espérions." Dyspnea, however, was rapidly relieved, with complete recovery.

CASE 5—1941—Gumbiner and Cutler[14] observed successful clearing of a pneumothorax in a newborn infant following the needle withdrawal of air. They used a 21-gauge needle with a Luer syringe, inserting it into the third left interspace 1 cm. to the left of the sternal border and directed medially, parallel to the inferior surface of the sternum.

SUMMARY AND CONCLUSIONS

1. A check valve obstruction of the bronchus by a vegetal foreign body is reported in a child.

2. The increased pulmonary pressure secondary to a foreign body in the bronchus was aggravated by the straining during bronchoscopy. A rupture of the alveoli followed with the escape of air

along the blood vessels to the mediastinum, following which there was generalized extension of air over the whole body by way of the blood vessel sheaths. The rapid and continuous passage of air into the body tissues led to marked ballooning and extreme cyanosis.

3. Fatal termination was prevented by an emergency cervical mediastinotomy.

1863 David Whitney Building.

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LXXXVIII

BILATERAL PNEUMOTHORAX IN A TRACHEOTOMIZED INFANT

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On February 5, a tracheotomized eight-month-old boy was admitted to the service at the Massachusetts Memorial Hospital, with a diagnosis of laryngeal stenosis.

His tracheotomy had been performed six weeks previous to admission, in a distant hospital, because of acute streptococcal laryngitis. The streptococcal infection abated, and repeated attempts were made to plug the tracheotomy tube, only to find each time that there was little if any laryngeal airway present. He was sent to us then, with the dual problem of reestablishing the laryngeal airway and decannulation.

The baby entered the hospital at 6:00 p. m. and three hours later the tube became obstructed. The Resident removed the whole tube, but was unsuccessful in reinserting it. With the child in extremis, the tracheotomy wound was reopened by the Resident, and a tube placed in the trachea. Following this procedure the child was very restless and the breathing, while improved, was unsatisfactory. He was placed in an oxygen tent but his condition grew steadily worse, as evidenced by marked dyspnea and cyanosis with epigastric retraction and a temperature of 105° F.

X-ray films of the chest were taken which confirmed the physical signs of extensive bilateral pneumothorax.

Dr. John Strieder, visiting chest surgeon to the Hospital, inserted a needle first into the right pleura and removed 250 cc. of air, bringing the pressure from positive to negative, and then inserted the needle into the left pleura, releasing 175 cc. of air and bringing the pressure from positive to negative.

Following this aspiration, the child was much improved. The respirations dropped from 74 to 40 and breath sounds could be heard on auscultation.

The child remained in the hospital four and a half months, during which time the larynx was dilated on several occasions. He was discharged with his tracheotomy wound closed and a normal laryngeal airway.

The possible incidents capable of producing the bilateral pneumothorax in this infant may be listed as:

1. Direct injury to the pleural domes either during the tracheotomy or the unsuccessful attempts at reinserting the tracheotomy tube. It is well known that in obstructed expiration the lungs may rise above the clavicles.

2. Rupture of air from the alveoli into the interstitial tissues of the lung, with migration of the air to the mediastinum and rupture from there into the pleural spaces.

3. Perhaps the most probable, the strong inspiratory movements of the child, sucking air into the mediastinum with eventual rupture into both pleural spaces.

It should be restated that the prophylaxis for pneumothorax and mediastinal emphysema is the introduction of a bronchoscope prior to tracheotomy. By this simple procedure a struggling child is converted into a quiet one with an adequate airway. The increased intrathoracic pressure incidental to obstruction is thus relieved and the operation becomes peaceful and tranquil.

29 Bay State Road.

LXXXIX

TRACHEOPATHIA OSTEOPLASTICA

(Osteoma of the Trachea)

Report of A Case

Louis H. Clerf, M.D.

Philadelphia, Pa.

Bony tumors of the tracheobronchial tree are rare and commonly produce few or no clinical manifestations. A majority of the cases reported were discovered at autopsy. Several of the recently reported cases were diagnosed by bronchoscopy. In Hiebman's[1] review of the literature more than 70 cases of bony tumors of the trachea were recorded up to 1934.

The origin of bony tumors in the tracheobronchial tree is not known. Various hypotheses have been offered which regard it as an exostosis, a congenital anomaly, an alteration of the elastic tissue or as originating from connective tissue elements.[2] Besides ossification of the cartilages of the trachea there may be nodular submucosal bony outgrowths, often extensive and occasionally so independent as to be classed as true osteoma. These often are in connection with the cartilages and the perichondrium.

The tiny multiple bony masses which form in the trachea and roughen its surface usually are designated as tracheopathia osteoplastica. Ribbert and Mischaikoff,[3] who have described these ragged nodules and plates of bone growing in the submucosa of the trachea, found that most of them, like tracheal ecchondroses, are connected with the perichondrium of the rings. Their relationship to the tracheal rings appears quite definite since the party wall was free from growths in the case reported by Moersch and his associates[2] and in the two cases included in this report.

The symptoms commonly observed are those secondary to obstruction. In Van Loon's[4] case the expectoration of blood-streaked sputum was the outstanding symptom. The following cases of tracheopathia osteoplastica, one exhibiting signs and symptoms of

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From the Department of Laryngology and Broncho-Esophagology, Jefferson Hospital.

Fig. 1.—Drawing of endoscopic appearance of trachea showing countless cone-like elevations varying from 4 to 8 mm. in height and covered with mucosa. Although the posterior or party wall was free from tumors, these were not limited to the tracheal rings but also filled in the interannular spaces.

pulmonary suppuration with hemoptysis, the other, hemoptysis of unknown origin are reported.

REPORT OF CASES

CASE 1. Female, age 50 years, was studied in 1940 for cough, purulent expectoration, hemoptysis and intermittent dyspnea. The clinical and roentgenological findings suggested a pansinusitis and chronic suppurative bronchitis with very probably basal bronchiectasis.

At bronchoscopy, the trachea presented a nodular appearance which became more marked as the carina was approached. There also was narrowing of the lower end of the trachea and of the orifices of both main bronchi so that a 6 mm. bronchoscope could not be passed beyond the carina. There was no ulceration noted. The posterior wall of the trachea was singularly free from nodules. A grating sensation was felt when the bronchoscope was passed into the narrowed portion of the airway. Considerable purulent secretion was observed coming from both bronchi. Several small nodules were removed with forward grasping forceps. These offered marked resistance and suggested calcareous or bony material. Histologic study of the material indicated that it was bone.

The patient has been observed at intervals during the past four years. The bony nodules do not appear to have increased in size. There are periods of fever, and at times wheezing and dyspnea are

Fig. 2.—Photomicrograph (x34.5) of bony tumor removed from tracheal wall. The surface is covered with squamous epithelium. Beneath this there are a number of spicules of bone in some of which a marrow cavity is developing. There also is present cartilage which is undergoing osseous changes. (Reported by Dr. C. J. Bucher).

distressing. There is definite evidence of bilateral basal bronchiectasis. A recent roentgen study of the trachea by Dr. Paul Swenson showed definite nodulation along the tracheal wall.

CASE 2. Male, age 45 years, was first observed in May 1943. There was a history of severe hemoptysis beginning eight years previously. This has occurred at irregular times and is unrelated to respiratory infections or other possible causes. On two occasions the blood loss was considerable. Repeated studies of the lungs, esophagus and stomach had failed to reveal any evidence of disease and studies of the blood were negative. No endoscopic examinations had been made.

On admission the patient was recovering from a recent severe hemorrhage, the sputum still was blood-tinged and blood was observed streaking the tracheal wall. Routine roentgen studies of the chest revealed nothing abnormal. At bronchoscopy there were observed innumerable small elevations which began above at the level of the first ring of the trachea continuing down to its bifurcation

and into the orifices of both main bronchi. The party wall was absolutely free from these and along the right side where the masses were more prominent the demarcation was very sharp (Fig. 1). There was narrowing of the trachea towards its lower end, also of both main bronchi. Considerable blood-tinged secretion was found present along the left lateral wall of the trachea suggesting that this might be a source of the hemoptysis. The nodular elevations were very firm and imparted a scratchy sensation to the bronchoscope. Several small masses were removed from the left wall of the trachea with forward grasping forceps for histologic examination. Histologic study revealed cartilaginous and bony tissue covered with squamous epithelium (Fig. 2). Planographic studies revealed an irregular pattern in the upper trachea suggesting a bony nodular surface. This could not be demonstrated in the lower trachea.

The management of this case posed a difficult problem. Although the interval between the attacks of hemoptysis was a number of months, the hemorrhage usually was severe. The extent of the process in the trachea precluded local endoscopic or surgical treatment. In consultation with Dr. Paul C. Swenson, Director of the Department of Radiology, it was decided to give a course of irradiation therapy. This was purely experimental, being given in dosage which would be entirely harmless but might be of value in arresting the process and also diminish the bleeding tendency. A total dosage not exceeding 750 roentgens was delivered into the lesion in the trachea.

The patient was examined nine months following the completion of the course of therapy. There was no apparent change noted in the tracheal lesions. While it probably is too early to evaluate the results of treatment, he has had no hemoptysis during the past nine months.

COMMENT

Although tracheopathia osteoplastica is admittedly of rare occurrence it should be considered as a possible cause of obscure hemoptysis and of tracheal obstruction. Irradiation is suggested as a possible plan of therapy. With a more general use of bronchoscopy in the study of patients with unexplained hemoptysis this condition may be more often encountered.

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XC

ADENOMA OF BRONCHUS TO MIDDLE LOBE, TREATED WITH ELECTROCOAGULATION

FREDERICK T. HILL, M.D.

WATERVILLE, MAINE

Adenoma of the bronchus is not an especially rare tumor. Clerf[1] reported 35 cases observed in 15 years, as compared to 243 cases of bronchial carcinoma. Undoubtedly many are never diagnosed, but, coming under medical care during periods of acute pulmonary infection, are considered as pneumonia. Recurrent attacks of pneumonia, localized to the same portion of the lung, should suggest the possibility of an obstructing tumor. Hemoptysis of bright blood, rather than rusty sputum, should likewise make one consider some other condition than pneumonia. Too frequently patients are discharged upon the subsidence of symptoms as cured of pneumonia without a final x-ray examination, when the persistence of roentgen findings might lead the physician to suspect an underlying cause of the condition.

This case is reported because of the initial difficulties in establishing the diagnosis and because of the apparently satisfactory response to coagulation therapy. I am emphasizing the qualifying adverb, realizing that the problem of treatment is far from settled. We all recognize that only if the tumor is endobronchial can anything be accomplished by coagulation; that these tumors grow very slowly and often tend to recur, sometimes years later; and that there may be a much more significant extrabronchial tumor which would require surgical extirpation.

REPORT OF A CASE

A young woman, 25 years of age, was referred to me for a diagnostic bronchoscopy, with a question of bronchial carcinoma. She had been admitted on the medical service with an acute pulmonary infection, fever, cough and bloody sputum. Clinical and x-ray findings were confined to the right middle lobe. While in the hospital

Read before the American Broncho-Esophagological Association, New York, N. Y., June 6, 1944.

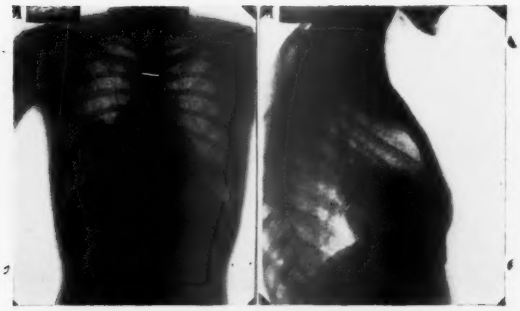

Fig. 1.—Roentgenogram made at previous hospital admission with diagnosis of pneumonia.

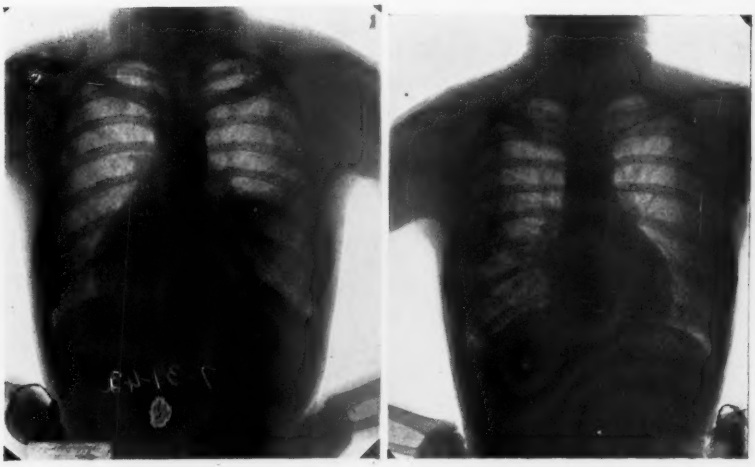

Fig. 2.—Roentgenogram on admission to hospital showing involvement of right middle lobe.

Fig. 3.—Lipiodal film showing obstruction in middle lobe bronchus.

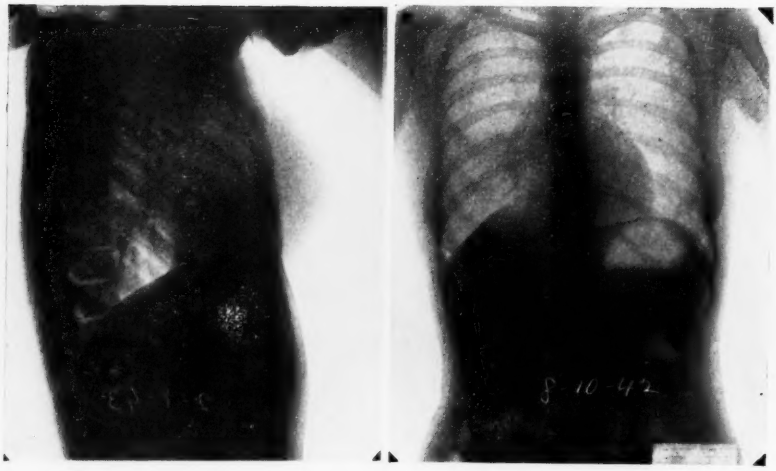

Fig. 4.—Lateral view showing obstructing tumor in bronchus to middle lobe.

Fig. 5.—Roentgenogram three months after completion of electrocoagulation.

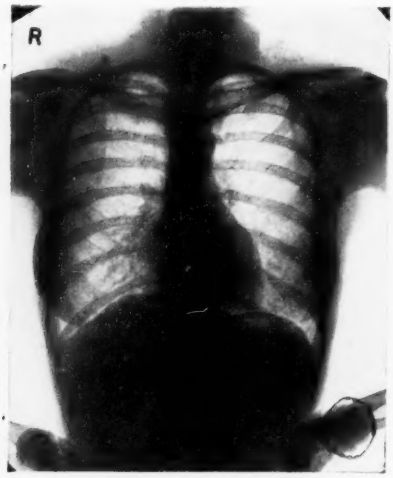

Fig. 6.—Roentgenogram one year after completion of treatment.

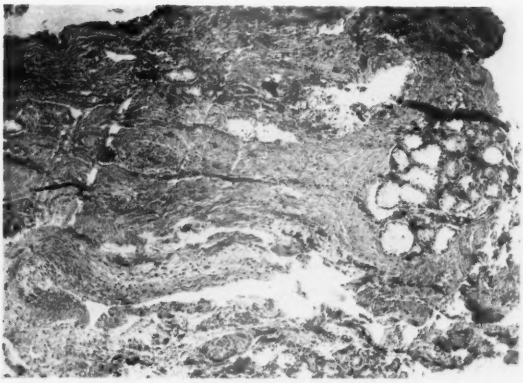

Fig. 7.—Photomicrograph of adenoma of bronchus. x53.

she had had a severe hemoptysis. Sputum was repeatedly negative for tubercular bacilli. After the acute symptoms subsided, roentgenograms showed a persistence of opacity in the middle lobe. There was a history of three attacks of pneumonia during the preceding year. This was especially significant when, on obtaining the chest films from the hospital where she had been treated previously, it was found that the location of the lesion had always been the middle lobe.

The first bronchoscopy resulted in apparently normal findings, but an attempt to put lipiodol into the middle lobe was unsuccessful. A second bronchoscopy, a few days later, revealed secretion coming from the orifice of the middle lobe bronchus but the cause of the obstruction could not be seen. A second lipiodol film showed partial filling and outlined what seemed to be a tumor just beyond the orifice. A third bronchoscopy, using a 5 mm. bronchoscope, showed a pink, slightly raised, soft tumor mass on the mesial wall a few millimeters beyond the orifice. Biopsy was followed by bleeding and two days later, while awaiting the histopathological report, she had a very severe hemoptysis.

Dr. Julius Gottlieb's report on the specimen was, "Chronic inflammation and an adenomatous hyperplasia of glandular epithelium suggesting a diagnosis of bronchial adenoma."

The tumor was then coagulated, using a 5 mm. Kernan coagulating bronchoscope. This was repeated in one week, and twice more at two-week intervals. Subsequently bronchoscopic examination

showed the tumor mass had disappeared and there was a small white scar at its former site on the mesial wall. X-ray films with lipiodol showed good filling and subsequent chest films have been considered as satisfactory. She has been symptom-free and able to resume her work for over a year now.

This is not reported as a cure but as a so-far satisfactory response to conservative therapy. The patient is under observation from time to time, and as long as she is symptom-free and chest films are satisfactory, elects to follow what seems to us, under the circumstances, to be the logical course. The location of the adenoma within the secondary bronchi added to the difficulties of diagnosis.

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XCI

DOES CHRONIC SINUSITIS CAUSE BRONCHIECTASIS?

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There are in the literature numerous articles supporting the ideas that sinus infections cause bronchiectasis. My clinical experience does not permit me to agree with this oft-quoted statement. The frequent coincidence of two diseases in the same patient does not prove that one caused the other, but rather suggests that both infections have the same etiologic factors. In discussions of this subject, mention is seldom made of the large number of patients who have had chronic suppurative sinusitis for years without developing cough or other bronchopulmonary symptoms. In an attempt to evaluate the relationship of these diseases, I have analysed the case histories of 50 patients who have bronchiectasis, and the case histories of 50 patients with chronic suppurative sinusitis of at least one year's duration who had no bronchopulmonary symptoms.

This is a controversial question. In order to eliminate misunderstanding, I shall attempt to define clearly the basis of the selection of cases, the criteria for accuracy of diagnosis, and the meaning of the terms used. The diagnosis of bronchiectasis was accepted only when confirmed by bronchograms made with iodized oil. The diagnosis of chronic sinusitis was accepted only if the patient had a history of purulent nasal and postnasal discharge of at least one year's duration, and if purulent sinusitis could be demonstrated.

The word allergy will be used frequently. This is a word with a very elastic meaning, so I think it is proper to define the meaning of the word as it is used in this discussion. The following brief definition by Zinsser, Enders and Fothergill[1] gives a clear picture of the basic concept of allergy as it applies to this discussion.

"Hypersensitiveness is an individual increased specific reaction-capacity to a substance, which in *normal* subjects of the same species, produces little or no reaction."

The inference is that a hypersensitive or allergic person reacts in an abnormal manner. In this discussion I hope to make it clear

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that, by reacting in an abnormal manner to the acute bronchopulmonary infections of childhood, the hypersensitive individuals develop bronchiectasis far more frequently than do the nonallergic members of the human species.[1] The clinical diagnosis of allergy in these cases was based on the presence of polyps in the nose or on the presence of many eosinophiles in the nasal and bronchial exudate.

Edema occurs in all local manifestations of hypersensitiveness. Therefore it does not seem surprising that bronchial obstruction due to edema, at the time of an acute bronchopulmonary infection, should very frequently lead to the development of bronchiectasis in allergic individuals.

In order to throw some light on the relationship of bronchiectasis, allergy and sinusitis, I have analyzed 50 cases of bronchiectasis in the manner shown in Table 1.

TABLE 1.—DATA IN 50 CASES OF BRONCHIECTASIS

LOBES INVOLVED	BRONCHIECTASIS	ALLERGY	SINUSITIS
Both lower lobes	14	12—85%	10—71%
Right lower lobe	28	23—82%	20—71%
Left lower lobe	8	5—62%	3—37%
TOTALS	50	40—80%	33—66%

Please note that 80 per cent of these bronchiectatic patients were hypersensitive and 66 per cent of them had sinusitis. These figures correlate closely with those of Watson and Kibler,[2] who in 1939 reported allergic manifestations in 82.6 per cent and sinusitis in 67.3 per cent of their cases of bronchiectasis. Diamond and Van Loon[3] in 1942 reported sinusitis clinically and roentgenographically demonstrable in 64 per cent of 75 bronchiectatic children. Walsh and Meyer[4] in 1938 reported sinusitis in 66.5 per cent of 217 bronchiectatic patients and Perry and King[5] found sinusitis in 66 per cent of 216 cases of bronchiectasis.

It is notable that all 33 patients who had both sinusitis and bronchiectasis were allergic. This, to my mind, indicates that the excessive mucosal edema found in hypersensitive individuals predisposes them to the development of chronic sinusitis, as well as bronchiectasis. Thirty-one of the 33 patients with sinusitis had a pansinusitis, as shown by x-ray examination. I think this diffuse bilateral involvement of the sinuses indicates a systemic or constitutional eti-

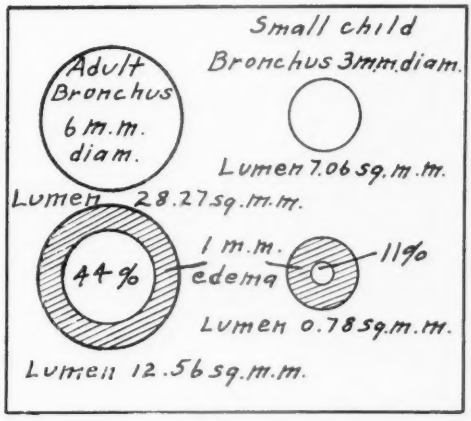

Fig. 1.

ologic factor such as hypersensitiveness. All 31 patients were in the hypersensitive group.

The idea that allergy is a factor in the development of bronchiectasis is not new. Watson and Kibler[6] in 1938 found allergic manifestations in the great majority of their patients with bronchiectasis.

If we are to prove or disprove this factor we must agree on what are the clinical signs and symptoms of the hypersensitive state and then look carefully for them in all cases of bronchiectasis. Few authors writing on this subject have taken the trouble to do so. For example, Lisa and Rosenblatt[7] in a monograph entitled "Bronchiectasis" published in 1943, dismiss the question by saying that this theory is not compatible with the pathological findings.

Andrus,[8] Holinger[9] and many others have shown that atelectasis and bronchial obstruction are the precursors of bronchiectasis. Friedman and Molony [10] have demonstrated the frequent occurrence of atelectasis in allergic children, so in the light of available evidence one cannot lightly dismiss the allergic factor in the pathogenesis of many cases of bronchiectasis.

The incidence of hypersensitiveness in the general population is, fortunately, not over 10 per cent. The incidence of allergic manifestations in 80 per cent of this group of 50 patients having bronchiectasis certainly suggests that allergy may be one factor in its pathogenesis.

Thirty-six of these 50 cases of bronchiectasis dated their chronic cough from a severe bronchopulmonary infection in childhood. The fact that mucosal edema in childhood has a marked effect in narrowing the lumen of a bronchus is well illustrated by Fig. 1. Please note that mucosal edema of only 1 mm. thickness reduces the lumen of a 6 mm. bronchus to 44 per cent of its normal area and that the same amount of edema in a 3 mm. bronchus reduces the lumen to 11 per cent of its normal area. To my mind this simple principle of geometry explains why atelectasis and resultant bronchiectasis develop so frequently in childhood in hypersensitive individuals. This figure, I think, also explains the preponderance of bronchiectasis in the left lower lobe because the bronchi on the left are narrower than the corresponding bronchi on the right.

Table 2 indicates the etiology of bronchiectasis as given in the history of each case.

TABLE 2.—ETIOLOGY OF BRONCHIECTASIS FROM HISTORY

Pneumonia	30
Whooping Cough	3
Diphtheria	2
Measles	2
Foreign Body	2
Following operation	2
Unknown	9

Table 3 indicates the data gleaned from the study of 50 patients who had chronic sinusitis but who did not have any bronchopulmonary symptoms. It has no bearing on the etiology of bronchiectasis but calls attention to the fact that many patients have pus running down the back of their throats for years, without developing any chronic bronchopulmonary disease.

TABLE 3.—50 CASES OF CHRONIC SUPPURATIVE SINUSITIS OF AT LEAST ONE YEAR'S DURATION

AVERAGE DURATION—6 YEARS

	SINUSITIS	ALLERGY
Nasal Origin	27	19—70%
Dental Origin	23	3—13%

Hoerr and Dixon[11] have offered convincing evidence, based on experimental work, that lymphatic drainage from the sinuses does not reach the lungs.

I include this series of 50 cases of chronic suppurative sinusitis to indicate that the presence of purulent post-nasal discharge does not invariably lead to bronchopulmonary infection, even in hypersensitive individuals.

There are thousands of children who each year simultaneously develop acute sinusitis and bronchopneumonia. It is my impression, based on clinical observation and the study of this series of cases, that the hypersensitive individuals frequently fail to recover completely from these acute infections and have as sequels chronic sinusitis and bronchiectasis.

Bronchoscopic aspiration is of unquestionable value in removing viscid obstructing exudate, but aspiration cannot remove edema of the bronchial mucosa. These remarks are not intended to minimize the importance of bronchoscopic aspiration in the prevention of bronchiectasis, but are intended to indicate that we should utilize additional therapeutic measures directed at reducing the mucosal edema at the time of the acute bronchopulmonary infection. Ephedrine by oral or subcutaneous administration does help to control mucosal edema during acute respiratory infections in hypersensitive individuals but better methods are desirable.

This paper will, no doubt, have some significance when we learn what allergy is and how to treat it adequately.

CONCLUSIONS

1. Chronic sinusitis and bronchiectasis frequently coexist, but one does not cause the other.

2. All patients in this series of cases who had both bronchiectasis and sinusitis were of the hypersensitive type.

3. The allergic or hypersensitive state was an etiologic factor in 80 per cent of this series of 50 cases of bronchiectasis.

4. Seventy-two per cent of these 50 patients developed their bronchiectasis during the first decade of life.

5. Seventy-four per cent traced the onset of their chronic cough to an acute bronchopulmonary infection.

6. Mucosal edema is probably the chief factor responsible for the atelectasis which led to the development of bronchiectasis.

7. Our results in the prevention and treatment of bronchiectasis will be better when we know more about hypersensitiveness and how to control it.

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Society Proceedings

SIXTY-SIXTH ANNUAL MEETING

OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

New York, N. Y., June 7-8, 1944

THE PRESIDENT, DR. CHARLES J. IMPERATORI, PRESIDING

(Abstracts of Proceedings)

Mucocele in Frontal and Ethmoidal Sinuses: Simplified Surgical Treatment

H. M. GOODYEAR, M.D.

CINCINNATI, OHIO

(This paper appeared in full in the June issue, page 242.)

DISCUSSION

Dr. Francis L. Weille asked if the speaker had had any experience with multilocular mucoceles.

Dr. Ralph Fenton questioned the practicability of this procedure in the case of a large mucocele extending over the roof of the orbit and often occupying a large part of the frontal sinus. He felt that such a case would be better taken care of through the external approach.

Dr. George B. Wood asked whether, from the etiologic standpoint, one should consider a mucocele as a cystic dilatation of some mucous gland or the result of occlusion of the duct, it being important to know whether the mucocele might be present without completely filling the sinus cavity.

Dr. Harris P. Mosher agreed with Dr. Fenton on the advisability of opening the frontal sinus in cases where it was invaded by the

mucocele. Also, he agreed with the essayist that nature's epithelialization of the cavity was far superior to any lining which surgery could produce. Dr. Mosher also pointed out the extreme size these growths might attain, by citing two recent cases in which exophthalmos and displacement of the bulb were marked and in which he felt that the frontal sinus was to a large degree involved.

Dr. Goodyear, in closing, remarked that he had never seen a multilocular mucocele, although he assumed such a condition might exist. As a rule the intranasal approach does not permit a full view of the entire cystic cavity.

If the external approach cannot be used he felt that it is not wise to remove the lining which nature had provided, and he would therefore be opposed to dissecting out the sac.

Commenting on Dr. Wood's question, Dr. Goodyear felt that the mucocele has its origin in a superior or anterior ethmoid cell in which the mucosa undergoes changes whereby the glands expand, and by pressure extend into adjacent cells and the frontal sinus.

Despite Dr. Fenton's and Dr. Mosher's comment, the author felt that if the mucocele is adequately opened intranasally and the passage kept patent, one will frequently find that the external deformity will quickly disappear. At any rate, he has been able to reduce it by simple pressure on the thin distended orbital plate.

The Local Use of Sulfadiazine, Radon, Tyrothricin, and Penicillin in Otolaryngology

S. J. Crowe, M.D.

Baltimore, Md.

(In the absence of Dr. Crowe his paper was read by Dr. Donald F. Proctor.)

(This paper appeared in full in the June issue, page 227.)

DISCUSSION

Dr. Thomas E. Carmody asked what amount of radium was used, since only the duration of the treatment was mentioned. Also, he questioned whether cessation of symptoms after one irrigation

could properly be called a cure, since he had seen the same result in years past regardless of what irrigating agent was used.

Dr. Howard Ballenger reported good results from the use of powdered sulfathiazole in acute nasopharyngitis. It has the advantage of remaining in contact with the tissues for a longer period than the solution and therefore need not be applied so often.

Dr. John McLaurin stated he had used radium and radon in small capsules threaded back through the end of a nasal cannula, but doubted whether this procedure would destroy all of the lymphoid tissure. Careful examination has convinced him that islands of this tissue persist even after irradiation, and continue to act as foci of infection. To destroy these completely might at the same time cause damage to normal tissue which has a definite value in the resistance against future colds.

To Dr. Harold I. Lillie's question whether controls had been used against the author's series, Dr. Proctor replied that they had.

Dr. DeForest C. Jarvis described his experience with apple penicillin in mother of vinegar, which can be given by mouth, applied locally, or by vaporization. He has seen striking results from its use in infections in livestock. Also, he has found it very effective in the treatment of auditory canal furuncles, eczema, hay fever (by inhalation), streptococcus sore throat and acute rhinitis. He recalled that the Duke of Wellington had given vinegar to his soldiers as a source of vigor and, long before that, the Roman legions used vinegar to prevent fevers and putrefaction.

Dr. Bernard J. McMahon inquired how long the treatment is continued and whether, following the use of penicillin, there is any tendency to recurrence of these upper respiratory infections.

Dr. Proctor, in closing, described a new type of radium applicator now being used under Dr. Crowe's supervision, which is made of monel metal, is smaller in diameter than the original model and is capable of holding 50 mg. of the salt. The dosage of one treatment is 5.5 mg. hours, i. e., 50 mg. for six and one-half minutes on each side, with 1 mm. of mass filtration which filters out the beta rays. The treatments are given once every four to six weeks over a long period of time. The average number of treatments for the ordinary child at Hopkins has been 3.4 per child, although in one case, despite 9 treatments, a large amount of lymphoid tissue was still present. Dr. Proctor stated that in most cases the lymphoid tissue can be elimi-

nated and claimed to have demonstrated this fact in hundreds of cases. However, if there is an infection in the tonsils or sinuses, radium therapy will be ineffective unless the focus is removed.

Although they have used sulfonamide powders a lot, they believe they get better results from the sulfadiazine solution which penetrates the mucosa, whereas the powder does not. As for a single irrigation curing an acute sinusitis, the essayist stated that these cases are followed up and irrigated again to make sure. After radical surgery in frontal or maxillary sinuses, irrigations are continued for a week or ten days and later at longer intervals, until the solution persistently returns clear.

Replying to Dr. Burt Shurly's question, Dr. Proctor stated that the tyrothricin was used in a 1:1000 alcoholic solution of which 1 cc. was diluted with 100 cc. of sterile distilled water. At operation, penicillin is used with equal parts of water.

Traumatic Deformities of the Nasal Septum

SAMUEL SALINGER, M.D.

CHICAGO, ILL.

(This paper appeared in full in the June issue, page 274.)

DISCUSSION

Dr. Thomas E. Carmody asked whether, in the Metzenbaum technique, the essayist ever found it necessary to remove some cartilage in order to get it into the groove of the vomer.

Dr. Salinger, in closing, stated that he appreciated the fact that the procedures depicted were subject to modifications at the hands of various surgeons to meet specific indications.

There are times when it is necessary to remove a part of the obstructing cartilage, but the important objective is the preservation of sufficient cartilage in the lower deviated portion to sustain the tip of the nose.

Teaching of Otolaryngology in Wartime

CAPTAIN HARRY P. SCHENCK, M.C., U.S.N.R.

OCEANSIDE, CALIF.

(Captain Schenck's paper was read by title.)

(This paper appeared in full in the June issue, page 221.)

Aerosinusitis: A Résumé

LT. COL. P. A. CAMPBELL, M.C.

RANDOLPH FIELD, TEXAS

High altitude aerial operations and the use of altitude chambers in the oxygen indoctrination program have led to an increase in the incidence and importance of aerosinusitis. Aerosinusitis results from a pressure differential between the inside and the outside of a sinus following environmental barometric pressure change, most often descent. Of the various complications of aerosinusitis, mucosal hematoma is probably of most importance. Histologically, these lesions consist of an accumulation of blood and fibrin between the loose tissues of the periosteum and those of the mucosa. They may be demonstrated roentgenographically. After a period of a few weeks to a few months hematomas in most cases resolve. Recovery from aerosinusitis usually follows equalization of pressure between the inside of the sinus and the external environment, regardless of the method by which equalization is accomplished.

DISCUSSION

Dr. Ralph Fenton expressed appreciation of Dr. Campbell's research in aviation medicine and the benefit that would ultimately accrue to civilian medicine as it has thus far in the service. He has noted the discomfort complained of by civilians who are forced to travel by plane at considerable heights in crossing mountain ranges, and the danger of complications, or at least exacerbation, among those suffering from sinusitis. Dr. Fenton inquired of Dr. Campbell whether the use of oxygen is of any particular value in obviating disagreeable results of altitude flying.

Dr. Robert S. Ridpath asked whether the essayist had noted any increase in the subjective symptoms during ascent or descent, particularly as to headache, pain or vertigo.

Dr. Thomas C. Galloway mentioned the work done at Northwestern University by Professor Ivy and Dr. Greenwood, with regard to the therapeutic effect of negative pressure in patients with acute respiratory disease and sinusitis. Gratifying results are reported in more than 50 per cent of the cases. These results would seem to be at variance with the effects observed by Col. Campbell, although Dr. Galloway admitted that in the Northwestern experiments the "descent" in the pressure chamber was accomplished at a much lower rate than was the case in the instances cited by the Colonel.

He also would like to know why there seems to be a difference between the Army and Navy requirements for fliers in so far as the status of the septum is concerned.

Dr. M. Martyn Kafka cited his experience with test pilots at a commercial airplant who ascend to high altitudes and frequently descend at a rapid rate. He has observed intense pain in the cheek and forehead in such cases. In one instance, a pilot who had an alveolar fistula was given relief by shrinking the mucosa about the opening. From this experience, he concluded that it might be a good idea to shrink the nasal mucosa just before a particularly trying flight was undertaken.

In the case of a man with bronchial asthma who was cautioned against going above 18,000 feet, the pilot reported having exceeded this altitude and, with the use of oxygen, having experienced no untoward effects. This fact was mentioned because of army regulations which ground any man with bronchial asthma.

The speaker mentioned his own work with the pressure chamber at Columbia University, and cited one instance of a man with a four plus deviated septum who was able to go as high as 47,000 feet without any unusual symptoms after preliminary shrinking of the mucosa with Neo-synephrin.

Dr. Rosenberg compared the effects of mass suction as frequently practiced in office treatment of sinusitis with those described by the essayist. The results of Col. Campbell's research would seem to be a good argument against such a practice.

Dr. Samuel Salinger felt that Col. Campbell's presentation was a clear exposition of a definite clinical entity. Considering predisposing causes, he mentioned the possibility of the size of the sinus having something to do with the incidence of hemorrhages such as the essayist described. The slides shown would seem to bear this out.

Comparing aerosinusitis with aerotitis, it is interesting to note that in the former we lack a simple prophylactic measure which is available in the latter; namely, autoventilation by the Valsalva procedure, excepting vasoconstriction which was mentioned.

In closing, Col. Campbell acknowledged the valuable assistance in this work which was made available by various physiological units in the field, and the interest and help afforded by interested colleagues. Replying to Dr. Fenton's query, he had found the use of oxygen of little value in the prevention of this complication. Aerosinusitis is the result of a combination of factors which must operate in the right sequence to cause the condition described.

As to aerial flight causing an aggravation of existing sinusitis, the author was quite sure that this was true, and consequently persons known to have such a condition had better avoid flying.

Concerning the Northwestern experiments mentioned by Dr. Galloway, the essayist stated that people feel exhilarated at varying altitudes, and it might be possible that the therapeutic effects claimed could be the result of the anoxia incident to altitude.

He did not believe septum resection aided many of these cases. It would be indicated only in cases where impingement of the septum on the turbinate causes blockage of the ostium.

Concerning the certainty of diagnosing hematoma, in his cases he is convinced that such was the case, since these conditions could be reproduced experimentally in dogs.

As to the relationship of the capacity of the sinus to the incidence of hemorrhage, the essayist agreed with Dr. Salinger. Aerosinusitis was observed most of the time in patients with large sinuses.

Summary of Some Known Facts Concerning the Common Cold

A. C. Hilding, M.D.

Duluth, Minn.

(This paper appeared in full in the September issue, page 444.)

DISCUSSION

Dr. George B. Wood agreed that a cold in its early stages is a virus infection and that it is urgent that we try to prevent the effects of secondary bacterial invasion. He demonstrated a practical method of obtaining cultures from the nose without danger of contamination. Commenting on the question as to the cause of the secondary bacterial activity, Dr. Wood was not sure whether it could be due to reduced local resistance or to some reaction between the virus and the bacteria. He recalled some experiments he had done on dogs some years ago at the University of Pennsylvania, in which infection could be induced only after the sinus mucosa had been mechanically irritated.

Dr. H. Marshall Taylor mentioned the effects of chilling without exercise and with exercise, the former being followed by a leukopenia, whereas the latter is accompanied by a leukocytosis. It would appear that exercise which increases respiration might be the cause of a rise in the temperature of the nasal mucosa, thus increasing its resistance to infection.

Dr. Samuel Salinger mentioned two items which might have a bearing on the predisposition to colds, one being the influence of a relatively dry mucosa and the other the question of native constitution. There is a difference in the vasomotor reactibility between the sympathetic and the parasympathetic types of individuals, which can be determined by a variety of procedures.

Dr. Arthur Proetz asked whether Dr. Hilding thought that individuals susceptible to chilling were generally in the hypothyroid group. Is there any connection between people who constantly feel chilly and those who are susceptible to chilling?

Dr. Hilding, in closing, cited the work of Paul and Frees in their studies of this problem up in Spitsbergen. They found that the natives isolated from the world for seven months were free from colds until the first boat came in the spring; then 90 per cent of the people developed colds. A second round of colds developed in the

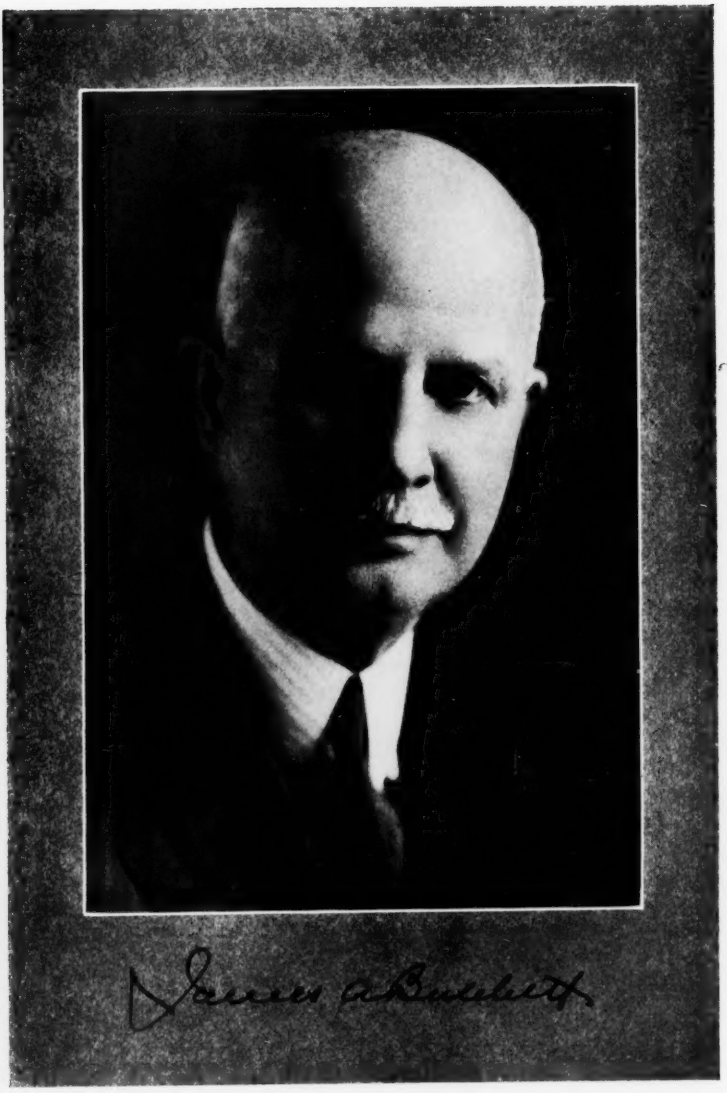
summer and then the incidence dwindled in the fall after the shipping season closed. They interpreted this to indicate that the cold virus was short-lived and dies out in a community unless new carriers come in.

Dr. Hilding stated he could corroborate Dr. Wood's experiments relative to the importance of trauma as a factor in the development of infection.

As to exercise and the possible raising of the temperature in the mucosa, he could not express an opinion, although he did know that in the normal individual exposed to chilling the depressed temperature did not last more than ten minutes.

He agreed that drying of the mucosa was a predisposing factor. Also, he felt that there might be something in the theory of sympathetic-parasympathetic constitution as factors, and would suggest it as a topic for further research.

As to the question of hypothyroidism in its relationship to cold susceptibility, he was unable to express any authoritative opinion.



JAMES A. BABBITT, M.D.

1869-1944

Dr. James A. Babbitt died on October 15, 1944, at the Lankenau Hospital, Philadelphia, Pennsylvania, of cerebral hemorrhage. In his death American otolaryngology has lost one of its most beloved, versatile, and active practitioners.

Dr. Babbitt was born in Waitesfield, Vermont, the son of the Reverend James Howard and Mary French Abbott Babbitt, and received his early education at the Phillips Andover Academy, graduating in 1889. He received his A.B. degree from Yale in 1893. From there he went to Haverford College to become Professor of Hygiene and Physical Education and to conduct classes in anatomy and bacteriology. He received his A.M. degree from Haverford in 1896. When he resigned from the Haverford faculty, he was elected Emeritus Professor.

While still carrying on his professorial work at Haverford he attended classes at the Pennsylvania Medical School from which he obtained his Doctorate of Medicine in 1898. His early training in otolaryngology was largely acquired in the clinics of the old Polyclinic Hospital under the tutelage of such men as Packard, Gibb, Vansant, Freeman and Watson.

During the earlier part of his career he was actively engaged in various phases of intercollegiate athletics. For six years he was president of the Middle Atlantic States Collegiate Athletic Association, a member of the National Football Rules Committee and Chairman of the Central Board of Officials. He is credited with the introduction of the game of soccer to this continent, and was responsible for bringing basketball to the State of Pennsylvania, the first game of the latter being played by the students of Haverford College. His interest in the athletic program of the country was very real but gradually his medical work, which was of course his chief interest in life, forced him to give up most of his activity in the athletic field.

To his chosen specialty he brought all of the tremendous energy and vitality that was so characteristic of his whole life. It is not necessary to note all of Dr. Babbitt's early hospital and teaching activities. Suffice it to mention that at his death he was Consulting Laryngologist to the Lankenau, Children's, Mary Drexel, Misericordia,

Fitzgerald Mercy, and the University of Pennsylvania Hospitals, and that he was on the courtesy staffs of the Byrn Mawr, Chestnut Hill, Episcopal and Presbyterian Hospitals. That his standing and ability as an otolaryngologist were nationally recognized is shown by his election to official positions in almost all of the national societies. He was past president and secretary of the American Laryngological Association, past president of the American Academy of Ophthalmology and the American Laryngological, Rhinological and Otological Society; past chairman of the Section on Otolaryngology of the College of Physicians of Philadelphia; member of the Board of Governors of the American College of Surgeons; fellow of the Otological Society and the Philadelphia Laryngological Association. He was Emeritus Professor of Clinical Otolaryngology at his Alma Mater and Associate Professor of Otolaryngology at the Graduate School of Medicine of the University of Pennsylvania.

During World War I he served in France and Germany as a Major in the hospital service of the American Red Cross.

Dr. Babbitt was brought up as a member of the Congregational Church but at the time of his activity at Haverford College he became an associate member of the Haverford Quaker Meeting.

His first wife, Mary A. Adams, died in 1911. Later, he married Marcella Hardwick Cordray who survives him. He is also survived by three daughters of his first marriage and a stepson and daughter.

Dr. Babbitt ranked as one of the leading otolaryngologists of this country and deservedly so. He not only had a natural aptitude for the acquisition of knowledge and the ability to use and apply this knowledge, but he also had an indefatigable zeal which sometimes pushed him to the utmost of his strength though he had unusual technique in the organization and regulation of his work.

He was a prolific writer and his contributions to otolaryngology covered almost the whole field of his specialty. He did, however, show a special interest in otology and in later years was deeply concerned with the problems of the hard of hearing.

He had a very kindly disposition, delighted in the friendship of his fellows and seldom if ever was he heard to utter a word of unkind criticism. Next to the satisfaction that came to him from his professional life was the joy he found in meeting with his fellow otolaryngologists at their various annual gatherings, and to their organization and programs he gave greatly of his time and energy.

Dr. Babbitt belonged to the University Club of Philadelphia, the Union League and the Merion Cricket Club. As a member of the Doctor's Golf Club he spent many happy hours on golf greens with his medical associates. He was also a member of the Society of the Mayflower Descendents, of Phi Beta Kappa and of Sigma Psi. The out-of-doors attracted him and his vacations were very apt to be spent on the salmon streams of New Brunswick.

Dr. Babbitt lived a life of usefulness and earnest endeavor. He gained a deserved national recognition. He was beloved by his patients, friends and medical comrades; he lived a life worth while. In his death American otolaryngology suffers a grievious loss and the memory of his comradeship and sterling worth will long remain in the hearts of its fellows.

GEORGE B. WOOD.

Abstracts of Current Articles

NOSE

Lysozyme in the Mucosa of the Nasal and Paranasal Sinuses.

Cabezas, J., Vaccaro, H., and Gonzalez, A.: Rev. de. Otorinolaryng. 4:37 (June) 1944.

The authors report an experimental study of the lysozyme content of normal and diseased mucosa of the nose and paranasal sinuses and its action on the saprophytic and pathogenic organisms most commonly found in these areas.

Forty-one patients were examined in this study. Mucus was taken from the turbinates and middle meatuses. The lysozyme content was variable in the different patients, and its activity was greatest in secretions obtained from the middle turbinate. The inhibitory power of lysozyme was shown to be present in solutions of 1 to 250 after 24 hours contact with Sarcina. In solutions of 1 to 80 the action apparently was immediate. Sarcina was used as the standard material for the control but other organisms, including staphylococcus, streptococcus viridans, and streptococcus hemolyticus were similarly affected, but no inhibition was produced on the pneumococcus.

In allergic rhinitis the presence of lysozyme appeared in such quantities that its inhibitory action appeared in solutions as low as 1 to 1000 after 12 hours contact. It is emphasized that these cases show a greater amount of lysozyme than either normal or infected patients, and that allergic secretions are sterile on direct culture.

The inhibitory properties of lysozyme are greatest in the secretions from the mucosa of the various sinuses, thus indicating that it has considerable to do with keeping these cavities sterile. In infected sinuses the lysozyme content in the secretion of the sinus mucosa was diminished more than 50 per cent for Sarcina and to an even greater extent for the ordinary pathogenic bacteria.

HIGBEE.

PHARYNX

Radium Treatment of Nasopharyngeal Lymphoid Hypertrophy.

Fricke, R. E., and Brown, H. A.: South. M. J. 37:399 (July) 1944.

In corroboration of the works of Crowe and his associates in the use of radium to reduce hypertrophied lymphoid tissue in the nasopharynx, the authors cite the following results:

Seventy-six patients were given nasopharyngeal radon irradiations.

Sixteen received two or more applications, with permanent improvement in 69%, temporary improvement in 6% and no improvement in 25%.

Sixty received only one application, with permanent improvement in 57%, temporary improvement in 20%, and no improvement in 23%.

The 61 patients who complained mainly of deafness and tinnitus showed definite and permanent improvement in 54%, temporary improvement in 18% and no improvement in 28%. Fifteen patients complained chiefly of frequent colds and sore throat, and of these 80% were apparently permanently improved, 13% temporarily improved and 7% not improved. In 15 of the 76 patients, the tonsils and adenoids were removed immediately before or after irradiation without changing the results of treatment.

A straight metal rod was used, containing radon (50 millicuries) filtered through 0.5 mm. of silver and 1 mm. brass. This was inserted through the nose into the nasopharynx and left in place for 30 to 60 minutes, administering a dosage of 1.5 to 3.0 gram minutes. Better results were obtained with the larger doses. The ages of the patients ranged from 2 to 48 years.

While the entire series was treated over a period of three years, the elapsed time between the treatment and the interpretation of the above results in individual cases was not stated.

McMAHON.

Lymphosarcoma of the Tonsil.

Kaplan, B.: Revista de Otorrinolaringologia, 3:222 (March) 1944.

In this article the author describes three cases of lymphosarcoma occurring in his practice. One early case is entirely well after three

and one-half years, one late case was fatal, and in another, prognosis is poor.

He makes the following general statements as to diagnosis and treatment. Lymphosarcoma of the tonsil is the most frequent tumor of the mouth and pharynx. Males are more often affected than females, and it is most frequent between the ages of forty and sixty. Pain is not a frequent symptom and when present occurs only late in the disease. The tumor is very sensitive to x-ray therapy, and the prognosis is good in those cases which show no metastasis along the cervical chain when first seen.

The tumor must be differentiated from simple hypertrophy, Hodgkin's disease, leukemia, benign tumors, syphilis, and agranulocytosis.

HIGBEE.

Tonsillectomy and Poliomyelitis.

Page, John R.: Arch. Otolaryng. 38:323 (April) 1944.

Page reports an incidence of one case of poliomyelitis, type unknown, occurring one month after tonsillectomy, in a series of 8,915 cases of tonsillectomy performed at the Manhatten Eye, Ear and Throat Hospital, New York, from 1937 to 1941, inclusive. During this period there occurred in New York, 941 cases of poliomyelitis, types unspecified, with 56 deaths.

MCMAHON.

LARYNX

Self-Inflicted Division of Larynx and Thyroid and Division of Trachea and Esophagus, with Recovery.

Schuessler, Willard W.: J. A. M. A. 125:551 (June 24) 1944.

This patient, using a straight razor, had made a transverse incision at the level of his hyoid bone, placed his fingers in his trachea and excised his hyoid bone, larynx, thyroid gland (including three parathyroids), his trachea down to about the third ring, about two inches of the anterior part of the esophagus, and about one-half inch of the posterior part of the esophagus.

Immediate gastrostomy was done and steps were taken by the surgeon to close the posterior wall of the esophagus and isolate the

trachea. Three weeks later the plastic repair of the esophagus was started by shifting flaps of skin from the anterior portion of the neck. Before leaving the hospital the patient was eating regular army rations and had gained 35 pounds.

McMAHON.

EAR

Bacteriological, Clinical, and Therapeutic Study of Acute Suppurative Otitis Media in Infancy.

Latorre, A. Augusto, and Landa, P. Francisco: Rev. de Otorinolaryng. 3:63 (Sept.) 1943.

The authors report a clinical and bacteriological study of 95 cases of acute suppurative otitis media in infancy. A comparison is made between a group of 45 in which sulfonamide therapy was used and a control group of 50 which received no sulfonamide therapy. Otherwise the two groups received identical treatment, which consisted of early myringotomy, heat, and irrigations.

Analysis of the two groups showed that sulfonamide therapy did not shorten the period of discharge or disability, and that in each group about the same number of cases became chronic or required mastoidectomy. Only two cases are stated to have required mastoidectomy, but 12 cases became chronic.

The bacteriological cultures showed: streptococcus hemolyticus in 59.3%; staphylococcus, 25%; pneumococcus, 22.3%; pneumococcus, 22.3%; and pneumococcus type 3 7.9%.

These authors discourage the use of the sulfonamides but urge early myringotomy and consider small blood transfusions of great importance.

HIGBEE.

Head Noises in Normal and in Disordered Ears—Significance, Measurement, Differentiation and Treatment.

Fowler, E. P.: Arch. Otolaryng. 39:498-503 (Mar.) 1944.

The writer states that tinnitus is present in 80 per cent of aural disease, though it may be absent in very severe and distressing disorders of the ear. He divides tinnitus into vibratory and nonvibratory types. The former are caused by local muscular contractions

and vascular irregularities about the head, neck and chest, and the latter by biochemical and drug irritations of the neural mechanism, as well as being associated with certain psychoses. He states that the therapy of head noises has been unsatisfactory mainly because attention has been focused too much on the symptom and not enough on its cause and on its place of origin. To afford the patient relief, either partial or complete, the condition should not only be treated locally, but the patient be treated generally and psychologically as well. The usual drugs advocated for relief of the condition have proven inefficient in his hands. Nicotinic acid and certain sedatives have been helpful in reestablishing the confidence of the patient, his sense of well being and a more healthful emotional response.

McMAHON.

The Industrial Noise Hazard.

McCoy, David A.: *Arch Otolaryng.* 39:327 (April) 1944.

McCoy obtained audiograms of 100 workers in a shipyard and selected those for observation, whose hearing appeared to be normal. At the end of one month audiograms were repeated and there was found a decided deficiency of hearing in the high frequencies in all cases. This deficiency was even more extensive at the end of one year. He stresses the need of aural protection, which can be obtained by placing noisy motors and machinery inside acoustically treated sound-proof rooms and observed and operated from the outside. He mentions the protection afforded by cotton or rubber stoppers placed in the ears, and comments upon the difficulty encountered in persuading workers to wear any type of ear protector consistently, because of the discomfort and lack of belief in their necessity. He feels that not much progress can be made in correcting such conditions unless executives are made fully aware of the aural hazards in certain types of industry, and will take the proper measures to ameliorate them.

McMAHON.

The Value of the Axial Projection of the Petrous Bone in the Diagnosis of Chronic Mastoiditis and Cholesteatoma

Danelius, Gerhard: *Radiology* 43:492-498 (Nov.) 1944.

The author describes the technique for obtaining radiographs by Ernest G. Mayer's method. Anatomical features are depicted in

two skull preparations. Different types and degrees of chronic inflammatory changes at the mastoid antrum are illustrated on cases corroborated at operation. Differential diagnostic limitations are discussed.

JORSTAD.

MISCELLANEOUS

The Use of Vaccines for the Common Cold; Status Report

Council on Pharmacy and Chemistry and Council on Industrial Health: *J. A. M. A.* 126:895 (Dec. 2) 1944.

This is a survey of controlled experiments with oral, parenteral and other types of vaccines reported by many authors in the literature of the past six or eight years. The evidence is carefully weighed and the following conclusions are reached:

"Vaccines prepared from a variety of bacteria commonly found in the respiratory tract have been prepared and combined in sundry ways and have been administered by various routes with the purpose of preventing colds, decreasing their incidence, ameliorating their symptoms, shortening their duration or decreasing their complications, or all of these combined. These objectives are all highly desirable. Unfortunately the evidence of individual case reports does not have any value in a disorder such as 'the common cold,' which probably covers a multiplicity of infections which are only symptomatically related. The only evidence which has scientific value is that which can be obtained by carefully controlled studies by qualified observers on large numbers of persons over a sufficiently long period of time to overcome the natural fluctuations in the major features of this symptom complex. Decisive evidence of the value of any vaccine is not forthcoming, and the weight of careful studies clearly indicates that none of the vaccines now available when administered by the routes advised have proved value. Vaccines for colds cannot be recommended for routine administration to industrial groups or to individuals. At present any attempt to prevent colds by the use of vaccines must be recognized as purely experimental and any proposal to administer such a vaccine, if given at all, should take this into consideration. As in all measures of a purely experimental nature, the uncontrolled use of any cold vaccine now available should be discouraged. Industrial physicians are under particular obligation to employ cold vaccines, if at all, only under the most rigidly controlled conditions and to report their results so that useless preparations can be promptly eliminated and further progress made."

Osteomyelitis of the Frontal Bone.

Otte, J.: *Rev. de Otorinolaryng.* 4:4 (June) 1944.

A study of twelve cases of osteomyelitis of the frontal bone is reported in which seven are referred to as localized, two as spreading, and three as fulminating. The seven localized cases are divided in respect to etiology as follows: Two are referred to as primary,

two followed measles, one followed scarlet fever, one was produced by trauma, and one followed a cold.

It is stated that none of these patients had sinusitis. Symptoms of acute infection such as high temperature, localized edema, and pain were present. The author states that the acute stage is followed by a period of quiescence which may last a variable period of time from weeks to even years. During this time only localized edema is to be found. The disease localizes in the diploic spaces producing necrosis and sequestra.

It is considered that necrosis of the mucosa follows that of its underlying bone for the reason that none of the patients showed signs of sinusitis during operation. During the quiescent period some patients showed extensions involving the dura and giving rise to dural abscesses; others showed extensions extending externally and producing inflammatory abscesses of the scalp.

In two cases the osteomyelitis followed chronic frontal sinusitis. When osteomyelitis is diagnosed, it is advised that Moser's technique of wide resection beyond the area be followed.

In three fulminating cases he distinguished two types of metastases—one a venous type which gives rise to bone necrosis without pus and in which infected areas appear at some distance from the original focus, and another type which extends directly through the diploic spaces.

Previous to operation the infection should be localized by the use of sulfonamides and transfusions.

HIGBEE.

Clinicopathologic Studies of Renal Damage Due to Sulfonamide Compounds. A Report of Fourteen Cases.

Murphy, F. D., Kuzma, J. F., Polley, T. Z., and Grill, J.: Arch. Int. Med., 73: 433-443 (June) 1944.

The cases reported in this paper and the comment on the subject are interesting to all physicians who use these drugs. It is pointed out that the damage to the kidneys is of two main types: (1) mechanical from precipitated crystals, and (2) direct toxic effect on the renal tissue, especially the tubular epithelium. There was no correlation between the blood level of the sulfonamide drug and the occurrence of renal damage. Chief warning signs of such injury were oliguria, anuria, hematuria, and azotemia.

HARFORD.

Oculo-Orbital Complications of Sinusitis.

Ameriso, Jose: *Revista de Otorrinolaringologia*, 3:213 (March) 1944.

When a case of unilateral or even bilateral cellulitis of the orbit is encountered, one should always consider infection of one or more of the nasal accessory sinuses as the etiologic factor.

About two-thirds of the orbit is separated from the paranasal sinuses by fragile bony walls. Numerous perforations in these walls which convey blood and lymph vessels and nerves are the portals through which the infection reaches the orbit.

The author states that the orbital periosteum is much less firmly attached than that of the sinus. For this reason, an infection which penetrates the sinus periosteum and bony wall may push the orbital periosteum ahead of it and create what he calls an inter-osteo-periosteal abscess.

Sphenoiditis is especially apt to cause blindness because of the extreme sensitiveness of the optic nerve to adjacent inflammation.

When the ophthalmic ganglion is involved, exophthalmos follows accompanied by an absence of tears which induces corneal ulcers and may terminate in panophthalmitis.

Enophthalmos frequently follows orbital cellulitis, because orbital fatty tissue resists infection very poorly and is replaced by scar tissue.

If the eyeball is infected by way of the lymphatics or blood vessels, iritis and retinochoroiditis occur.

External incision and drainage is urgent in those cases which show involvement of the optic nerve or meninges. In less severe cases, surgery is contingent upon the results of general supportive care and medication.

HIGBEE.

Notices

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

A sum of money having accrued from the Casselberry Fund of the American Laryngological Association, a prize will be offered in 1945 for original investigation in the art and science of laryngology or rhinology. Theses must be in the hands of the Secretary, Dr. Arthur W. Proetz, 1010 Beaumont Building, St. Louis 8, Missouri, before March 1, 1945.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct examinations on the following dates:

June 5-8, 1945, Hotel Waldorf-Astoria, New York, New York.

October 3-6, 1945, Palmer House, Chicago, Illinois.

All communications should be addressed to the Secretary, Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.

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OF THE

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Meeting: Palmer House, Chicago, October 7-11, 1945.

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Sections:

Eastern—Chairman: Dr. Oram R. Kline, 414 Cooper St., Camden, N. J.
Meeting: Benjamin Franklin Hotel, Philadelphia, January 12, 1945.
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Meeting: Charlotte Hotel, Charlotte, N. C., January 15, 1945.
Middle—Chairman: Dr. Carl H. McCaskey, 608 Guaranty Bldg., Indianapolis, Ind.
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Western—Chairman: Dr. Aubrey G. Rawlins, 384 Post St., San Francisco, Cal.
Meeting: Elks Club, Los Angeles, January 27-28, 1945.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Meeting: New York, June 11-15, 1945.

American Otological Society

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